

EXHIBIT 37

IN THE UNITED STATES DISTRICT COURT
DISTRICT OF NEW JERSEY

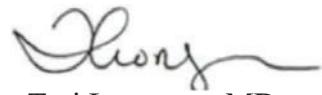
IN RE JOHNSON & JOHNSON
TALCUM POWDER PRODUCTS
MARKETING, SALES PRACTICES, AND
PRODUCTS LIABILITY LITIGATION

MDL NO. 16-2738

THIS DOCUMENT RELATES TO:
Newsome, et al. v. Johnson & Johnson, et al.
3:18-cv-17146

**RULE 26 EXPERT REPORT OF
TERI LONGACRE, MD**

Date: May 28, 2024



Teri Longacre, MD

I. BACKGROUND AND QUALIFICATIONS

I am a board-certified diagnostic surgical pathologist at Stanford Medicine with subspecialty expertise in gynecologic pathology. I received my medical degree in 1985 from the University of New Mexico School of Medicine in Albuquerque, New Mexico, where I completed my residency training in anatomic and clinical pathology. Following residency, I completed a fellowship in surgical pathology at Stanford University. Thereafter, I took a position as Assistant Professor at Stanford University and rose through the ranks of the professoriate to my current position as Professor of Pathology. I am the Emerita Richard L. Kempson Endowed Chair in Surgical Pathology at Stanford University School of Medicine, where I serve as Director of Gynecologic Pathology and Director of the ACGME-approved fellowship in Gynecologic Pathology, a program I founded in 2007. In addition, I am the Director of Gastrointestinal Pathology and Director of the ACGME-approved fellowship in Gastrointestinal Pathology, a program I also founded in 2013. I am the former Director of the Stanford Hospital Tissue Committee and a former member of the Stanford Care Improvement Committee, which oversees the quality of patient care in the hospital. In addition to many other extramural committee appointments, I am a former President of the Association of Directors of Anatomic and Surgical Pathology, in part due to my prior work as a Director of Surgical Pathology at Stanford.

I have internationally recognized expertise in benign and cancerous conditions of the female reproductive system, including cancers of the ovary, uterus, cervix, vagina and vulva, and have published extensively in the peer-reviewed medical literature on gynecologic pathology. I provide continuing medical educational lectures on gynecologic pathology to practicing pathologists regionally, nationally and internationally, and have authored and co-authored numerous review articles, book chapters and a textbook in gynecologic pathology. I also provide annual resident and fellow lectures at Stanford Medicine in the areas of non-neoplastic and neoplastic gynecologic pathology and examine gynecologic pathology specimens, including ovarian cancer specimens, on a routine basis. I have published and lectured extensively on the topic of ovarian cancer pathology. Because of my expertise in gynecologic pathology, I was invited to become a member of the American Board of Pathology Test Committee, which provides gynecologic pathology questions for the certification exam for pathology residents and for the maintenance of certification exam for practicing pathologists. I co-authored the 4th edition of the World Health Organization (WHO) Breast and Gynecologic Tumours, and am an expert editor and co-author of the 5th edition of the WHO Classification of Tumours: Female Genital Tumours. I am also editor of the 7th edition of Sternberg's Diagnostic Surgical Pathology and associate editor of the chapters on gynecologic pathology in that book. I am a member of a number of pathology societies and editorial boards, a list of which is provided in the attached curriculum vitae (Exhibit A), which also sets out my education and training in detail and lists my peer-reviewed publications, committee appointments, invited lectures and active grant funding.

My clinical diagnostic activities chiefly include examination of surgical gynecologic and gastrointestinal specimens, including small biopsies and large organ resections. My annual case volume amounts to 5,000 to 7,500 cases; one-half to one-third are gynecological cases and of those, twenty to thirty percent are ovarian cancer cases. In addition to anatomic pathology, I am board certified in clinical pathology, which enables me to integrate findings in the areas of chemistry, hematology, microbiology, immunology, molecular pathology and other special laboratory studies as they relate to my practice of gynecologic pathology. In this capacity, I

routinely provide clinical and pathologic consultations to physicians at Stanford Medicine; this entails macroscopic (gross) and microscopic review of surgical pathology specimens and review of relevant clinical information to render informed patient diagnoses. I am a regular participant in the Stanford Gynecologic Oncology Interdisciplinary Tumor Board as well as several Gastrointestinal Tumor Boards. In addition to the clinical work I provide for Stanford patients, I also receive requests for my consultative opinion from both pathologists and treating physicians regionally, nationally, and internationally.

My opinions are held to a reasonable degree of medical and scientific certainty and are based on my education, training and experience, as well as my clinical and scientific research, general knowledge of the literature, my pathologic review of thousands of ovarian cancer cases throughout my career, and my review of the relevant medical records and pathology in this case. I reserve the right to amend or supplement my opinions, if additional, relevant information becomes available to me. The references and attached materials list (Exhibit B) include many sources that I have considered in forming my opinions; of course, it is impossible for me to identify here all sources of information I have considered over the many years of my career.

I am compensated at a rate of \$600 per hour for consulting on this case.

II. OVARIAN CANCER

Ovarian cancer is not a single disease. It comprises a set of distinct cancers, each of which exhibits different clinical, histological, epidemiological, and molecular underpinnings. Ovarian cancer can be separated into two broad groups: epithelial ovarian cancer and non-epithelial ovarian cancer (e.g., germ cell, sex-cord stromal tumors), as well as a variety of miscellaneous tumors and metastases. The plaintiffs' expert reports focus on epithelial ovarian cancer, and not any other type of ovarian cancer.

A. Epithelial Ovarian Cancer (EOC)

Epithelial ovarian cancer (EOC) is the most common type of ovarian cancer and is comprised of multiple, distinct diseases, including high-grade serous carcinoma (HGSC), low-grade serous carcinoma (LGSC), mucinous carcinoma, endometrioid carcinoma, clear cell carcinoma, and other rare subtypes.

HGSC is, by far, the most common type of EOC. These tumors arise from tubal-type epithelium, usually in the fallopian fimbria and, less commonly, on the ovarian surface or within ovarian epithelial inclusion cysts. For those that arise on the ovarian surface or within inclusion cysts, the current belief is that these result from deposition of fallopian tube epithelium that subsequently undergoes transformation at these sites. Nearly every HGSC, including precursor lesions, harbors a deleterious mutation in the *TP53* tumor suppressor gene, which is considered to be one of the earliest known molecular events in the development of HGSC (Ahmed 2010; Vang 2016). In addition, HGSC are deficient in homologous recombination and lack the ability to repair double strand DNA breaks. About 10% to 12% of women with HGSC carry germline mutations in the *BRCA1* or *BRCA2* genes. An additional, smaller percent of HGSC occur in association with germline mutations in other homologous recombination genes (Song 2014). Additional genes that have been associated with hereditary ovarian cancer include the tumor suppressor gene, *TP53*, in

the Li-Fraumeni syndrome, as well as several other genes involved in the double-strand breaks repair system, such as *CHEK2*, *RAD51C*, *RAD51D*, *RAD50*, *BARD1*, *BRIP1*, *MRE11A*, and *PALB2* (Madariaga 2019). Recent literature suggests that close to 25% of ovarian cancers are associated with germline mutations, and of these, 29% have mutations in genes other than *BRCA1* or *BRCA2*. (Frey 2017). Other patients may have a clear familial predisposition to developing ovarian carcinoma for as yet unknown reasons. The *BRCA1/2*, *CHEK2*, *RAD51*, *BRIP1*, *BARD1*, *MRE11A*, and *PALB2* genes encode proteins that play a critical role in maintaining genomic stability by promoting error-free DNA repair. (Gudmundsdottir 2006; Toss 2015). Approximately one-third of sporadic HGSC may also contain somatic *BRCA1* or *BRCA2* mutations, *BRCA1* methylation, or genomic aberrations in these other homologous recombination genes. (Madariaga 2019).

Ovarian cancers associated with homologous recombination defects exhibit a series of characteristic morphologies. Most are HGSC with marked nuclear pleomorphism with giant bizarre nuclei and high mitotic index (Fujiwara 2012; Soslow 2012). They also tend to show a solid, pseudoendometrioid, or transitional cell carcinoma-like (“SET”) morphology (Soslow 2012). When T-cell subtypes are examined, *BRCA1/2*-mutated tumors exhibit significantly increased CD3+ and CD8+ tumor infiltrating lymphocytes, as well as elevated expression of PD-1 and PD-L1 in tumor-associated immune cells compared to homologous recombination-proficient tumors. Women with *BRCA1/2*-mutated HGSC tend to have a better overall prognosis and response to chemotherapy (particularly PARP-inhibitors) than women with non *BRCA1/2*-mutated HGSC (Dann 2012).

It is now well established that a significant majority of so-called “ovarian” HGSC arise from the distal fimbrial end of the fallopian tube from a precursor lesion known as serous tubal intraepithelial carcinoma (STIC). Criteria for site-assignment in extrauterine HGSC have been proposed (Singh 2015; McCluggage 2015; Singh 2014; Singh 2016; Singh 2016b) and the use of these criteria result in a high-proportion of previously presumed “ovarian” HGSC (approximately 80%) being classified as tubal in origin, while primary peritoneal HGSC are exceedingly rare. A diagnosis of primary peritoneal HGSC should only be made when there is no ovarian parenchymal HGSC and no mucosal STIC or HGSC within either tube, both of which should be grossly visible in their entirety and histologically examined in total using a SEE-FIM protocol.

Clinically, HGSC are aggressive tumors that primarily affect older women (median age at diagnosis is 63) (SEER Cancer Stat Facts: Ovarian Cancer, 2024). Most patients (75%) who develop HGSC present in advanced stage (FIGO III-IV), and there is no currently accepted approach to reduce mortality through early detection (Goff 2000; Hogg 2004). The 5-year survival of women with advanced stage disease is about 20% compared with 80-90% for those with FIGO stage I-II disease. While HGSC often responds to standard platinum-based chemotherapy, prevention strategies have largely been through surgery: prophylactic and risk-reduction salpingo-oophorectomy in women who are at elevated risk, such as those with germline *BRCA* mutations or strong family cancer histories (Kauff 2002). Prophylactic salpingo-oophorectomy reduces the risk of *BRCA*-related gynecologic cancer by 96% (Haber 2002). Although the risk of ovarian cancer is diminished, there remains a small risk (0.8-1%) of subsequently developing a peritoneal HGSC, especially in women who have mutations in the *BRCA1* and *BRCA2* genes (Casey 2005). Oral contraception use appears to reduce the risk of HGSC, as do number of term pregnancies and breastfeeding. The protective effects of these apparent diverse risk-reducing factors is attributed

to interruption of ovulation and hence, decreased proliferation of the tubal epithelial cells now believed to be involved in the development of HGSC. A recent study that demonstrated tubal ligation results in decreased proliferation of the progenitor cells in the distal fallopian tube corroborates this theory (Tiourin 2015).

HGSC is now believed to be distinct from low-grade serous carcinoma (LGSC). Except for their shared morphologic resemblance to tubal-type epithelial cells, HGSC and LGSC differ in genetic abnormalities, pathways of tumorigenesis and clinical behavior. Whereas HGSC is an aggressive tumor affecting older women, the clinical course of LGSC is typically more prolonged, with LGSC typically affecting women of somewhat younger age. LGSC is more refractory to chemotherapy than HGSC, probably because of the lower proliferative rate of the former. LGSC arises by step-wise progression from benign through borderline to malignant tumors, while most HGSC arises *de novo* in the fallopian tube. While HGSC commonly shows mutations in *TP53* and *BRCA1* or *BRCA2*, LGSC is more likely to have mutations in *BRAF*, *KRAS*, and *NRAS* (Romero 2020). Women with germline *BRCA* mutations are at increased risk of HGSC, but not of serous borderline tumor or LGSC, further underscoring differences in pathogenesis between HGSC and LGSC. LGSC is not associated with mutations in homologous recombination genes. The histologic distinction between HGSC and LGSC is based primarily on nuclear features, with less than three-fold variation in nuclear size in LGSC. A secondary diagnostic criterion is mitotic activity, LGSC having less than 12 MF/10 HPF. There are also differences in architectural features between LGSC and HGSC, with micropapillary architecture and psammoma bodies more common in the former, while HGSC frequently shows solid growth pattern, at least focally, which is an uncommon feature in LGSC. With the exception of p53, immunohistochemistry is not particularly helpful (and is seldom required) to separate LGSC and HGSC.

In contrast to HGSC, endometriosis is a well-recognized precursor of ovarian endometrioid carcinoma and clear cell carcinoma. At least 25% of ovarian endometrioid carcinomas will have foci of concomitant endometriosis on pathologic examination, while up to 47% of clear cell carcinomas have foci of concomitant ovarian endometriosis on pathologic examination and up to 68% have concomitant endometriosis in the pelvis (Fadare 2019). Accordingly, when endometriosis is found in the overall specimen, pathologists regard this as compelling evidence of an origin from endometriosis, even in the absence of a demonstrable transition from endometriosis. Endometriosis is commonly associated with adenomyosis in the uterus; an MRI study documented a prevalence of endometriosis in the setting of adenomyosis of 80.6% and a prevalence of adenomyosis in the setting of endometriosis of 91.1% (Zannoni 2020; Leyendecker 2015). Data from RNA transcriptional and DNA methylation analyses suggest differences in menstrual cell cycle state (e.g., proliferative or secretory) of endometrial progenitor cells may account for the different cellular phenotypes and clinical behaviors of endometrioid and clear cell carcinomas (Beddows 2024).

Endometrioid carcinomas exhibit morphologic features that resemble uterine endometrial carcinoma and are graded similarly (i.e., FIGO grade 1, 2 or 3), depending on the degree of glandular differentiation and the presence of cytologic atypia. Like their uterine counterparts, they often exhibit foci of squamous metaplasia. They harbor mutations similar to those seen in the uterine corpus (*PTEN*, *PIK3AC*, *CTTNB1*, *ARID1A*). Mutations in *TP53* are uncommon (no more than 11%-24%), particularly in the lower grade tumors, and they are not associated with genomic aberrations in homologous recombination genes. Endometrioid ovarian carcinomas often express

the receptors for estrogen and progesterone. Mismatch repair protein deficiency can be seen in almost 20% of tumors and 3%-10% harbor mutations in *POLE*. Up to 30% of ovarian endometrioid carcinomas are associated with endometrioid endometrial carcinoma (Romero 2020). A family history of ovarian endometrioid cancer in a first-degree relative, or any ovarian cancer, has been associated with an increased risk of ovarian endometrioid cancer, with relative risks that range from 2.81 to 3.81 (Fadare 2019).

Clear cell carcinomas have a distinct appearance characterized by clear or eosinophilic cells arranged in papillae, cysts, and solid nests. They are not graded. Like endometrioid carcinomas of the ovary, they are not associated with genomic aberrations in homologous recombination genes. Like ovarian endometrioid carcinoma, ovarian clear cell carcinoma may be associated with mutations in *ARID1A*, which is currently considered an early molecular event when these tumors arise from endometriosis. Other gene mutations include *PIK3AC*, *KRAS*, and *PPP2R1a*. Mutations in *TP53* are uncommon (no more than 20%) (Romero 2020). Clear cell carcinomas typically do not express receptors for estrogen and progesterone. Like ovarian endometrioid carcinoma, ovarian clear cell carcinoma may be associated with mutations in genes that encode DNA mismatch repair proteins (Bennett 2016). These mutations may be somatic or germline. Germline mutations in these genes are seen in patients with Lynch syndrome, a hereditary cancer syndrome that is associated with increased risk of developing carcinomas in the uterine corpus, ovary, colon, and renal pelvis, as well as a variety of other organs. Approximately 11% of all ovarian carcinomas arise in women with Lynch syndrome; most of these are endometrioid, clear cell, or undifferentiated.

Ovarian mucinous carcinoma is uncommon; because of the identification of a background teratoma or Brenner tumor in some cases, an ovarian teratoma or Brenner tumor has been considered a possible precursor (Simons 2020). Ovarian mucinous carcinoma is typically unilateral and low stage. Many of these tumors arise in the background of a mucinous borderline tumor. When recurrences occur, they do so early. Response to standard chemotherapy is poor. *HER2* amplification and overexpression is present in approximately 16% of cases, and targeted therapy against *HER2* has been proposed for use in these cases. Ovarian mucinous carcinoma is typically not associated with mutations in homologous recombination genes or DNA mismatch repair genes. The most common gene mutations are *CDKN2A* (76%), followed by *KRAS* (64%) and *TP53* (64%), and less commonly, *BRAF*, *PIK3CA* and *ARID1A* (Romero 2020).

Carcinosarcoma contains malignant epithelial and malignant mesenchymal elements, each of which are derived from the same clonal origin. The epithelial component is usually high grade and most often resembles serous or endometrioid carcinoma, but malignant mucinous, squamous, or clear cell elements or undifferentiated carcinoma, including small cell carcinoma of the pulmonary type, may be encountered as well. The mesenchymal component may have the features of a fibrosarcoma, leiomyosarcoma, endometrioid stromal sarcoma, or nonspecific sarcoma, or, in heterologous tumors, a rhabdomyosarcoma, chondrosarcoma, or osteosarcoma. Intracellular and extracellular hyaline droplets, which are PAS-positive, may be present in the sarcomatous and sometimes in the carcinomatous component. Carcinosarcomas develop most often in postmenopausal women (del Carmen 2012). They often harbor mutations in *TP53*.

Mesonephric-like carcinoma is a recently recognized tumor that exhibits morphological and immunophenotypic features suggestive of mesonephric adenocarcinoma, but is not anatomically associated with mesonephric remnants (Pors 2021). Ovarian mesonephric-like carcinomas exhibit

variable histologic patterns including glandular, tubular, papillary, and solid growth. Some cases show intraluminal eosinophilic colloid-like material. These tumors are diffusely positive for PAX8, but negative for ER and PR. GATA3 and TTF1 are both diffusely positive in these tumors. Mutations in *KRAS*, *NRAS*, *BRAF*, *CTNNB1* and/or *PTEN* have been identified in these tumors, supporting Mullerian epithelial derivation. They do not harbor mutations in *TP53*. Although data are limited, they appear to be clinically aggressive and are currently not graded.

Undifferentiated carcinoma exhibits no or only rare and minor foci of epithelial differentiation (Bennett 2021). Less than 5% of ovarian carcinomas are undifferentiated. A subset is associated with Lynch syndrome.

Mixed carcinomas account for less than 3% of ovarian carcinomas and most commonly consist of admixtures of endometrioid and clear cell carcinoma (Ye 2014)), often arising in association with endometriosis or endometrioid and undifferentiated carcinoma (so-called de-differentiated carcinoma).

In addition to the carcinomas described above, there exists a set of serous and mucinous borderline tumors in the ovary.

Serous borderline tumors (SBT) account for the vast majority of all ovarian borderline epithelial neoplasms and comprise approximately 15% of all ovarian serous neoplasms (Vang 2020). SBT is encountered most often between the ages of 30 and 60 years, whereas serous carcinomas are most common between the ages of 40 and 70 years. SBT is typically bilateral and has the capacity for extra-ovarian spread, recurrence, and death, even though the tempo of disease progression is significantly more indolent when compared to LGSC. The molecular profile of mRNA gene expression patterns is significantly different for SBT and LGSC versus HGSC (Gilks 1998), as is the pattern of genetic alterations, e.g., the presence of point mutations in *BRAF* or *KRAS* is more frequently associated with SBT and LGSC, while *TP53* mutation and somatic or germline abnormalities in *BRCA1* and/or *BRCA2* are more frequently associated with HGSC. SBT are composed of architecturally complex branching papillary and micropapillary structures not unlike that of LGSC, but they do not feature destructive invasion of the ovarian stroma. The nuclei are uniform or mildly atypical and mitotic activity is low. Atypical mitotic figures are absent. Transformation to LGSC occurs in at least 7% of women with SBT, occasionally decades after initial diagnosis. Transformation is associated with increased tempo of disease and a significantly more aggressive disease course with approximately 40-50% overall survival. In some instances, transformation is preceded by several recurrences of SBT, which may or may not exhibit increasing degrees of atypical proliferation. In other cases, the transformation appears at the time of first recurrence. Most transformations occur in the omentum, followed by intraabdominal or axillary lymph nodes (Longacre 2005). Very rarely, SBT transforms to a high-grade carcinoma.

Mucinous borderline tumors are composed of enteric-type epithelium (gastric type cells, goblet cells, and occasionally Paneth cells). They occur in women in the fourth to seventh decades, are typically unilateral, and quite large (19 cm in average diameter). They are benign, provided they do not harbor foci of mucinous carcinoma (Talia 2022). They may harbor mutations in *KRAS*.

Seromucinous borderline tumors are distinguished from SBT by the presence of both serous and mucinous endocervical-like epithelial cells with abundant neutrophils, and the more frequent

association with endometriosis. Although these tumors may also be associated with peritoneal implants, no tumor-associated deaths have been reported, and the prognosis is excellent (Talia 2022).

Endometrioid borderline tumors are uncommon (Bell 2000). Up to 40% are associated with endometriosis. They may be bilateral or associated with synchronous endometrioid tumors in the endometrium and/or fallopian tube. Like their malignant counterpart, they are associated with mutations in *PTEN* and *CTTNB* (Oliva 2006) amongst others. They are benign.

B. Non-epithelial Ovarian Cancer

Non-epithelial ovarian cancer consists of tumors derived from the specialized ovarian stroma (sex cord-stromal tumors) and germ cells. Sex-cord stromal neoplasms account for approximately 6% of all ovarian tumors. They contain elements of sex cord and stromal derivation, either pure or in varying combinations. The most common subtype is the fibroma. The remainder exhibit differentiation toward one or more of the following cell types: granulosa cells (most frequent) and Sertoli and/or Leydig cells (least common).

Adult granulosa cell tumor typically occurs in postmenopausal women (Li 2022). Approximately 75% are associated with estrogenic signs and/or symptoms. Adult granulosa cell tumors are unilateral and considered to be of low malignant potential. They may recur decades after diagnosis. Prognosis depends on stage of disease at presentation. They are composed of ovoid cells with euchromatic or hypochromatic nuclei, often exhibiting a “coffee bean” appearance and small, central nucleoli. The cells are arranged in a variety of patterns that vary from solid, insular and trabecular to anastomosing cords. Both macrofollicular and microfollicular (Call-Exner bodies) patterns are often emphasized, but they are absent in the majority of tumors. The microfollicular pattern is characterized by small, generally regular follicles (Call-Exner bodies). Up to 97% of adult granulosa cell tumors harbor a mutation in *FOXL2*.

Juvenile granulosa cell tumor tends to occur in children and young adults (Young 2018). Occasionally, it is seen in older women. Most, but not all juvenile granulosa cell tumors are clinically benign. They are composed of sheets or nodular aggregates of round to ovoid cells surrounding follicles that vary in size and shape and often contain eosinophilic or basophilic secretions. Call-Exner bodies are rare. They are not associated with *FOXL2* mutations.

Sertoli-Leydig cell tumors comprise less than 0.5% of ovarian tumors; they arise from Sertoli stromal cells (Young 2018). These neoplasms occur most frequently in women less than 40 years old (median 28 years old) and may present with hormonal manifestations (androgenic or estrogenic). Tumors are typically unilateral. Well differentiated tumors are considered benign tumors and are not associated with recurrence. In contrast, moderately to poorly differentiated tumors are regarded as malignant neoplasms and have a 5-year survival of approximately 78%. They have a heterogeneous morphology composed of neoplastic Sertoli cells arranged in tubules, cords, trabeculae, or sheet-like architecture and scattered Leydig cells. Heterologous differentiation, most frequently as a gastrointestinal or rhabdomyosarcomatous component may be present, but may also include chondroid, smooth muscle, or neuroendocrine, especially in the moderately and poorly differentiated tumors. The prognosis of these tumors is primarily based on grade, stage and presence of heterologous elements. Most of the moderately and poorly

differentiated tumors are associated with somatic or germline mutations in *DICER1*. *DICER1* syndrome is a rare tumor predisposition disorder associated with genetic alterations in the *DICER1* gene located on chromosome 14q32.13. This syndrome presents in children and adolescents and confers an increased lifetime risk of a variety of benign and malignant neoplasms, including tumors of the lung (pleuropulmonary blastoma), gynecologic tract, thyroid (multinodular goiter, thyroid carcinoma), kidney (cystic nephroma, Wilms' tumor), head and neck (nasal chondromesenchymal hamartoma, ciliary body medulloepithelioma), central nervous system (pituitary blastoma, pineoblastoma), and various soft tissue sarcomas (Han 2022).

Microcystic stromal tumor is a rare ovarian neoplasm that occurs in adults (Young 2018). This tumor is composed of small cells arranged in a microcystic pattern masses separated by hyaline bands and fibrous plaques. The cells express nuclear beta-catenin. These tumors harbor a mutation in *CTNNB1* and may be an extracolonic manifestation of familial adenomatous polyposis.

Steroid cell tumors comprise approximately 0.1% of ovarian tumors and are composed of large round or polyhedral steroid cells (Liu 2005). They are divided into Leydig cell tumor and steroid cell tumor not otherwise specified (NOS). Leydig cell tumors arise in the hilus or less commonly, within the ovarian stroma (Leydig cell tumor, non-hilar type) in postmenopausal women, causing hirsutism or virilization in 80% of patients. Occasionally, there are estrogenic manifestations. These tumors are associated with an excellent prognosis. On the other hand, steroid cell tumors, not otherwise specified (NOS) may occur at any age and carry a risk for metastasis. Patients frequently present with virilization due to androgen excess (41%), but occasionally with estrogenic or no endocrine manifestations. In children they may induce isosexual pseudoprecocity or result in Cushing syndrome. The tumors are almost always unilateral and FIGO stage I but 20% of patients have extraovarian extension of their tumors at the time of diagnosis.

Germ cell tumors account for approximately 30% of all ovarian tumors (Euscher 2019). Most (95%) are mature cystic teratomas (dermoid cysts). The frequency of malignant germ cell tumors is higher in countries whose populations are largely Oriental or black, in whom ovarian epithelial carcinomas are relatively uncommon. Germ cell tumors account for two-thirds of ovarian cancers during the first two decades of life.

Mature teratomas, which are almost all cystic (dermoid cysts), account for approximately 25% of all ovarian tumors. These tumors usually develop in children or reproductive age women but are sometimes not detected until years after menopause. Rarely mature teratomas are familial. Mature solid teratomas are occasionally accompanied by mature glial implants, which are almost always associated with an excellent prognosis. Monodermal teratomas include struma (thyroid), carcinoid (neuroendocrine) and strumal carcinoid (thyroid and neuroendocrine), amongst other, rarer subtypes. Mature teratomas are benign, except for those that harbor adult-type malignancies (Euscher 2019).

Immature teratomas are the third most common primitive germ cell tumors, accounting for almost 20% of all cases (Euscher 2019). They are most often found in young adults and children (median, 18 years). One-third of immature teratomas are FIGO stage II or III. These tumors are diagnosed on the basis of immature neuroectodermal elements characterized by neuroepithelial rosettes and tubules or cellular foci of mitotically active glia. Almost 90% to 100% of patients respond to combination chemotherapy with sustained remission. Mature tissue may continue to grow

(growing teratoma syndrome), requiring a second operation. Patients with exclusively mature peritoneal implants, which are composed of glia, almost always have a benign clinical course, even in the absence of postoperative treatment.

Dysgerminomas are the most common of the primitive germ cell tumors; they account for nearly half of such tumors (Warnnissorn 2021). Eighty percent of dysgerminomas develop in women younger than 30 years of age and are rare over 50 years. In patients with associated gonadoblastoma (these tumors often contain areas of calcification), an underlying abnormality in gonadal development may or may not be clinically apparent. Dysgerminomas are often unilateral and low stage. They are malignant, but respond to chemotherapy and radiation therapy with an 80% 5-year survival rate for patients with higher stage or recurrent disease. Most tumors recur within the first 2 years, but occasionally recurrence is late, even beyond 10 years. The tumor cells resemble primordial germ cells and are arranged in a predominantly diffuse or alveolar or insular pattern separated by thin to broad collagen bands infiltrated by mature lymphocytes. More than 80% of dysgerminomas show chromosome 12p abnormalities, either as i(12p) or 12p overrepresentation. Approximately one-third have *C-KIT* mutations.

Yolk sac tumors comprise about 20% of primitive germ cell tumors of the ovary (Young 2022). They occur most frequently in childhood and adolescence (mean age, 19 years). Rarely, *somatic* yolk sac tumors may be associated with endometrioid, serous, or mucinous carcinomas or carcinosarcomas in postmenopausal patients. Serum alpha fetoprotein level is almost always elevated preoperatively. This is a rapidly growing, highly malignant neoplasm, and evidence of extraovarian spread is present in approximately one-third of patients. Response to combination chemotherapy is high. The prognosis is poor when somatic yolk sac tumors are associated with an epithelial ovarian carcinoma. The tumor is composed of primitive tumor cells with clear cytoplasm (due to glycogen content and, occasionally, lipid content) and hyperchromatic, irregularly shaped large nuclei arranged in reticular, microcystic or macrocystic patterns. Schiller-Duval bodies may be present in up to 75% of cases.

C. Miscellaneous Tumors

A variety of miscellaneous tumors arise in the ovary. Most notable types are small cell carcinoma, hypercalcemic type and tumors of probable Wolffian origin.

Small cell carcinoma, hypercalcemic type occurs in young females between 15 and 30 years, with a peak in the early 20s (Tischkowitz 2020). Approximately two-thirds of the tumors are associated with paraendocrine hypercalcemia. Small cell carcinomas are almost always unilateral, although involvement of the opposite ovary may be seen as part of the abdominal spread encountered at laparotomy in approximately a third of the cases. These are aggressive neoplasms with a poor prognosis, and the majority of patients die of their disease, usually within 2 years. They are composed of diffuse sheets of small, closely packed, round to occasionally spindle-shaped cells with scanty cytoplasm. Follicle-like structures lined by tumor cells are present in 80% of cases. These spaces typically contain eosinophilic, but occasionally basophilic, fluid. In 40% of tumors, a variable proportion of large cells have abundant eosinophilic cytoplasm. These tumors are associated with inactivating mutations of *SMARCA4*.

Tumors of probable Wolffian origin typically arise in the broad ligament, but may arise in the ovary (Shalaby 2020). They are composed of tumor cells that grow diffusely or form closely packed solid or hollow tubules. A sieve-like appearance is often present. The cysts contain eosinophilic luminal secretions. The tumor cells are oval or spindle shaped, and they have scanty eosinophilic cytoplasm or pale cytoplasm in solid tubular areas. They most often resemble endometrioid tumors of the ovary and are often mistaken for them. Most are benign, but occasional cases metastasize.

D. Metastases

Metastatic tumors to the ovary are common and may be misinterpreted as primary ovarian carcinomas, particularly if they arise in the gastrointestinal tract (Zhang 2020). Gastrointestinal tract primary carcinomas can exhibit features similar to primary endometrioid and mucinous adenocarcinomas and it can be difficult to distinguish them. Breast carcinoma may also metastasize to the ovaries; this can be particularly problematic in patients with *BRCA1/BRCA2* germline mutations.

In summary, ovarian carcinoma is not a single disease or even a single set of multiple diseases. The vast majority are epithelial “ovarian” cancers, but this group of tumors is composed of multiple tumor types, each with a different clinical presentation, different histopathology, different molecular pathogenesis, different disease course, and different response to various types of chemotherapy. The non-epithelial ovarian malignant tumors are similarly diverse, and there are several types of miscellaneous tumors that arise independent of both the epithelial and non-epithelial tumors. Moreover, the frequent occurrence of metastases to the ovaries and their misinterpretation as primary ovarian cancer that has historically clouded the classification of ovarian cancer continues to pose diagnostic and therapeutic difficulties to this day. Given this vast array of cancers that can occur in the ovary, it is inconceivable that any single endogenous or exogenous factor or factors can be attributed to their diverse etiologies.

III. TALC AND OVARIAN CANCER

Foreign material, however inert, will evoke a response in human tissue. The initial response can be associated with acute inflammation, involving local macrophages and mast cells, and may evolve into a foreign body reaction, with formation of multinucleated foreign body giant cells and granulomas that function to wall off the foreign material from the surrounding tissue. Talc is known to elicit a foreign body reaction in human tissue (Clement 2019; Shah 2017; Irving 2015; Reichert 2012; de Brito 1994; Mostafa 1985; Perou 1973). This host response to talc is exploited in talc pleurodesis, a common FDA-approved procedure for treatment of benign and malignant pleural effusions, as well as pneumothorax. Talc is currently considered the most effective sclerosant available for pleurodesis.

There is no correlation between the presence of talc and ovarian carcinoma. Although the early literature hypothesized a possible role of talc in the development of ovarian cancer (e.g., Cramer 1982; Henderson 1971, 1979), subsequent accumulated data from human and animal studies have not substantiated this link (e.g., O’Brien 2020; Taher 2019; Berge 2018; Penninkilampi 2018; Visvanathan 2018; Gonzalez 2016; Houghton 2014; Terry 2013; Gates 2010; Keskin 2009; Cramer 2007; Gertig 2000; Heller 1996; Boorman 1995). Studies reporting talc

particles in cancerous and non-cancerous tissue have been cited in support of this hypothesis; however, talc (like other small mineral particulates) is relatively ubiquitous, especially in the medical profession (Heller 1996; Henderson 1971, 1979; McDonald Mar 2019; McDonald Oct 2019; Campion 2018). Talc particles identified in excised patient tissues without associated foreign body reactions are more likely than not the result of post-surgical contamination from tissue processing; such findings cannot be reliably linked to the pathogenesis of an individual patient's ovarian cancer. Indeed, if talc-related inflammation was an inciting factor in the subsequent development of ovarian HGSC, one would expect to see talc-related foreign body responses associated with the early HGSC precursor lesions (STIC) (Clement 2019; Reichert 2012; Perou 1973). This is not seen. Nor are these early STIC lesions (with the exception of intratumoral lymphocytes) associated with inflammation or with reported talc use (Malmberg 2016; Visvanathan 2018).

Current evidence does not support chronic inflammation as a cause of ovarian cancer. HGSC, the most common type of ovarian cancer, is associated with STIC precursor lesions. STIC exhibits similar histologic features to that of HGSC. Both tumors may show papillary, solid, so-called pseudoendometrioid and transitional morphology. Cytological atypia is marked. Intratumoral lymphocytes may be present and can be numerous. However, there is almost never an associated chronic inflammatory process. No significant correlation has been demonstrated between HGSC and the histologic presence of chronic inflammation or chronic tubal injury (Malmberg 2016). Further, pelvic inflammatory disease, an inflammatory condition that affects the fallopian tubes and ovaries, is associated with gross and microscopic evidence of chronic inflammation and fallopian tube injury but is not reliably associated with the development of ovarian cancer (e.g., Huang 2021; Rasmussen 2017; Zhou 2017; Shen 2016). Inflammation has not been shown to be a driver of HGSC. Likewise, inflammation has not been shown to be a driver in the development of endometrioid or clear cell ovarian cancers, the next most common subtypes. Endometrioid and clear cell carcinoma are highly associated with endometriosis. Endometriosis is a multifactorial disease, and recent epidemiologic evidence suggests factors other than inflammation contribute to the development of endometriosis-associated ovarian cancers. (Huang 2021). Unlike most chronic inflammatory conditions, endometriosis-associated inflammation is hormonally driven. Defects in steroid hormone signaling contribute to the growth and survival of endometriotic tissue and these likely play a role in malignant transformation. (Bulun 2019). Several studies have demonstrated the presence of known cancer-driver mutations in uterine endometrium (*KRAS*, *PIK3CA*, *ARID1A*) that are also found in endometriotic lesions and endometriosis-associated ovarian cancers, and these likely play a role in the development of endometriosis and in ovarian cancer. (Bulun 2019). There is also some evidence to suggest that some women may have an inherited predisposition to endometriosis. (Bulun 2019). Although pelvic inflammatory disease has been tendered as a possible risk factor in ovarian cancer, the cumulative incidence rate of ovarian cancer is in fact significantly higher in patients with endometriosis than in those with pelvic inflammatory disease ($p < 0.001$) (Huang 2021).

Talc foreign body reactions are not associated with chronic, tissue destroying inflammation. Some chronic inflammatory conditions may be associated with risk for developing cancer. Inflammatory bowel disease (e.g., ulcerative colitis) and the development of colon cancer is one such example. Another example is that of squamous cell carcinoma arising in a chronic skin wound. Despite underlying differences in the etiology of the chronic inflammation, *each at its core is associated with histological evidence of long-term, chronic inflammation with tissue*

destruction. Talc foreign body reactions and granulomas are not associated with this type of chronic, tissue-destroying inflammation and have not been associated with an increased risk of cancer (Shah 2017; Hunt 2007; de Brito 1994).

Talc particles reported in human tissue, in the absence of biological reaction, are likely lab contaminant. Reported general findings of “birefringent” particles in tissue are irrelevant if the particles are not in the plane of section of the tissue involved by tumor, within macrophages or other cells, and/or if there is no associated inflammation or foreign body reaction in the areas or tissues in which the particles are located. Of note, many of the aggregates of birefringent material depicted in publications are sufficiently large that they would not be present *in vivo* without an associated foreign body response. In the absence of this expected response, one can only reasonably conclude that this material is contaminate introduced during or after surgery. If talc were present in tissue prior to resection and processing, smaller particles would be expected to be seen within macrophages, while larger particles would be expected to elicit a foreign body giant cell reaction – neither of which is demonstrated in publications. Moreover, claims in publications concerning talc in tissues of purported talc users (including some users who develop ovarian cancer) have not validated their claims with histopathologic evidence of exposure, and such findings are not consistent with my substantial experience as a gynecological pathologist and my examinations of thousands of tissue specimens from women with gynecologic cancers, including ovarian cancer.

Although publications recognize that “talc contamination of the surface of surgical pathology specimens is common,” (McDonald Mar 2019), these publications fail to recognize that laboratory processing of tissue specimens for histology can not only introduce contaminants on the surface of the specimen, but also deep within tissue (Heller 1996; McDonald Mar 2019). No published literature reports methods to adequately control for the tissue processing following surgery, making histopathologic correlation critical to support claims of biologic exposure. There are multiple steps in tissue processing following surgery that may (and often do) introduce particulate contaminate into the tissue. The fixation and processing of pathology specimens can result in introduction of foreign particulate throughout the specimen and not just on the surface of the tissue. The experienced diagnostic pathologist is well aware of the potential introduction of such material during tissue processing and refrains from issuing a diagnostic opinion in the absence of corroborating evidence of an associated cellular (i.e., presence of particulate material within macrophages with clear displacement of the macrophage cytoplasm) or foreign body reaction.

Migration studies that claim to demonstrate that talc migrates from the perineum are not compelling. To date, virtually all studies in humans have been based on the introduction of various materials directly into the vagina, cervical os, and/or uterus, and not on perineal exposure alone (e.g., De Boer 1972; Egli 1961; Iturralde 1981; Kunz 1996; McCalley 1985; Sjosten 2004; Ventner 1979). Aside from their questionable relevance to an individual’s use of perineal talc, these studies do not control for effects of body positioning (e.g., Trendelenberg), endogenous or exogenous exposure to oxytocin, anesthesia, or surgery (De Boer 1972; Egli 1961; Iturralde 1981; Kunz 1996; Kunz 2007; McCalley 1985; Ventner 1979). Similarly, studies that specifically utilize inert carbon particles do not control for random exposure to environmental carbon particles (Egli 1961; Wehner 1985). Also, animal studies, which are difficult to extrapolate to humans, have yielded conflicting results (Edelstam 1987; Keskin 2009; Phillips 1978; Thompson 2061). No study has provided conclusive evidence that talc, when applied to the perineum of the human female can penetrate the

cervical barrier and “migrate” to the fallopian tube and peritoneum in the absence of deliberate manipulation.

There is also no compelling scientific evidence that talc particles in tissue cause “ovarian” cancer. Studies reporting gene-talc interactions, immune-talc interactions, and interactions between talc and the oxidative system are largely correlative and have not been independently substantiated as bona fide mechanisms of ovarian carcinogenesis (Gates 2008; Fletcher Mar 2018; Saed 2017). The examination of cancerous and non-cancerous tissues from a patient with ovarian carcinoma with scanning electron microscopy and energy dispersive X-ray analyses is also not a sufficiently scientific or appropriate methodology for demonstrating a causal link between the presence of talc and/or asbestos and the development of the patient’s ovarian cancer (McDonald Mar 2019; McDonald Oct 2019; McDonald Nov 2019). At a minimum, a histologic response (e.g., foreign body reaction) in association with the presence of birefringent particles or the presence of particulate material within macrophages with clear displacement of the macrophage cytoplasm should be present to confirm actual exposure and to exclude artifact (e.g., Clement 2019; Reichert 2012; Perou 1973). In absence of this response, it is more likely than not that the particles are processing contaminants or “innocent bystanders.” Even in the presence of the expected histologic response, a convincing link between the presence of a foreign particle and carcinogenesis cannot be established using this methodology.

In summary, there is no scientific support to the claim that talc causes the varied and distinct diseases that are broadly referred to as “ovarian” cancer. Ovarian carcinoma is not a single disease. Although the majority are epithelial “ovarian” cancers, this diverse group of tumors is composed of multiple tumor types, each with a distinct clinical presentation, pathology, and molecular pathogenesis. No scientific study has linked talc exposure to the specific genetic alterations associated with development of all these different tumors and it is implausible that perineal exposure to talc provides a biologic mechanism for the development of these distinct diseases.

IV. CASE FINDINGS

In preparing my case-specific report, I reviewed the relevant medical records (e.g., operative, surgical pathology, and genetic testing reports) and available H&E stained slides from Tamara Newsome’s [REDACTED]

[REDACTED] In brief, Tamara Newsome is an African American, [REDACTED], with prior history of [REDACTED]. At the time of her diagnosis of ovarian endometrioid carcinoma she was 53 years of age. There is [REDACTED]

[REDACTED] The 31 H&E stained slides from her surgical procedure, which represent recuts prepared by Holy Cross Hospital, Silver Springs MD demonstrate low-grade endometrioid carcinoma (FIGO grade 1) involving the right ovary (Figure 1) and uterine serosa (Figure 2) (slides A4, A5, and A8) (FIGO stage IIA). The left ovary, right and left fallopian tubes, and omentum are uninvolved. No lymph node tissue is identified in the specimen designated as right pelvic lymph node. The carcinoma is associated with endometriosis (Figures 3 and 4) (slide A8) as well atypical endometriosis (Figures 5-7) (slides A13 and A15). Immunohistochemical stains performed on unstained slides labeled A8 in the CLIA laboratory at Stanford demonstrate the tumor cells are positive for PAX-8 (Figure 8) and cytokeratin 7 (Figure 9), but not cytokeratin

20 (Figure 10), which is compatible with ovarian endometrioid cancer (and not compatible with metastatic colorectal cancer). The tumor cells further demonstrate loss of MSH6, but not MSH2 or PMS2. CD10 highlights the stroma around the endometriotic gland (Figure 11). In addition, sections of the uterus demonstrate deep adenomyosis (Figure 12) (slides A6 and A7), cellular leiomyoma (slide A5), and benign, inactive endometrium. A *MUTYH* mutation of uncertain significance was identified on myRisk panel through Myriad.

Birefringent material is present in the slides reviewed. However, the absence of associated foreign body reaction (or even presence of particles in macrophages) identifies this material as artifact, most likely from the processing of the tissue for histology.¹ There is no chronic inflammation associated with the cancer.

V. RESPONSE TO PLAINTIFF'S EXPERT

Plaintiff's pathology expert, Dr. Godleski, asserts that there is "birefringent foreign material" in 8 of the 31 slides he reviewed. The composition of this material is unknown, as it was not subjected to further analysis by Dr. Godleski. The lower two photos of Figure 1 of his report include pictures purporting to show such birefringent material in "dense collagenous stroma" and "densely cellular ovarian stroma" in areas not involved by tumor. Although he states these particles are within the plane of focus, it appears that they are overlying the tissue and not within it as he asserts. The particles are not within cells and there is no associated inflammation in these areas.

Additional photos of birefringent material were produced separate from Dr. Godleski's expert report and were also reviewed. In many of the provided pictures, it is not clear that the observed birefringent material is in the same plane of section as the tissue. There is no associated foreign body reaction in Dr. Godleski's photographs and no definitive evidence of particulate in macrophages (Godleski Report, Figure 1, and additional photomicrographs produced by Plaintiff). In addition, many of the aggregates of birefringent material depicted by Plaintiff's expert are sufficiently large that they would not be present *in vivo* without an associated foreign body response. As noted above, if talc were present in tissue prior to resection and processing, smaller particles would be expected to be seen within macrophages, while larger particles would be expected to elicit a foreign body giant cell reaction – neither of which is demonstrated.

Although Dr. Godleski recognizes that "talc contamination of the surface of surgical pathology specimens is common" (McDonald Mar 2019), he fails to recognize that laboratory processing of tissue specimens for histology can not only introduce contaminants on the surface of the specimen, but also deep within tissue. Despite the report's arduous description of attempts to exclude the possibility of talc contaminant, Dr. Godleski did not control for the tissue processing following surgery. There are multiple steps in tissue processing following surgery that may (and often do) introduce particulate contaminant into the tissue. The fixation and processing of pathology specimens can result in introduction of contaminants throughout the specimen and not just on the surface of the tissue. The experienced diagnostic pathologist is well aware of these potential contaminants and refrains from issuing a diagnostic opinion in the absence of associated foreign

¹ To the extent asbestos is alleged to be a contaminant of talcum powder, I saw no evidence of ferruginous bodies in the available tissue to support exposure. Also, the reported association between asbestos exposure and ovarian cancer has been questioned (Slomovitz 2020; Reid 2011).

body reactions. I found no foreign body reactions supportive of talc exposure in the available slides and no evidence of particulate in macrophages.

Dr. Godleski also opines that “it can be stated to a reasonable degree of medical certainty, that the talc and tremolite asbestos found in the tissues of this case are contributory evidence for a causal link between the presence these materials and the development of this patient’s ovarian cancer.” (Godleski Report, 7). Examination of cancerous and non-cancerous tissues from a patient with endometrioid carcinoma with scanning electron microscopy and energy dispersive X-ray analyses is not a sufficiently scientific or appropriate methodology for demonstrating a causal link between the presence of talc and/or asbestos and the development of the patient’s ovarian cancer. At a minimum, a histologic response (e.g., foreign body reaction) in association with the presence of birefringent particles or the presence of particulate material within macrophages with clear displacement of the macrophage cytoplasm should be present to confirm actual exposure and to exclude artifact (e.g., Clement 2019, Reichert 2012; Perou 1973). In absence of this response, it is more likely than not that the particles are a contaminant or “innocent bystander”. Even in the presence of this histologic response, a convincing link between the presence of a foreign particle and carcinogenesis cannot be established using this methodology.

VI. SUMMARY OPINIONS

There is no reliable scientific basis to conclude that talc (or any component of talcum powder) is an etiologic factor in the pathogenesis of ovarian cancer. Although several studies have reported finding talc in ovarian tissue using light microscopy and ultrastructural analysis, none has validated their claims of exposure with the known and expected histopathologic findings associated with talc. Without histopathologic correlation, laboratory contamination/artifact cannot be excluded, and this is the most likely explanation for the reported findings. Further, examination of cancerous and non-cancerous tissues from a patient with endometrioid carcinoma with scanning electron microscopy and energy dispersive X-ray analyses is not a sufficiently scientifically appropriate methodology to demonstrate a causal link between the presence of talc and the development of the patient’s ovarian cancer. The evolution of the talc-ovarian cancer hypothesis is highly reminiscent of the evolution of the (historically incorrect) hypothesis that herpes simplex virus (HSV-2), a venereal transmitted virus, was causally associated with cervical cancer. This theory was advanced largely on the basis of seroepidemiological findings (higher prevalence of HSV-2 antibodies among cancers than controls), documented HSV infection and electron microscopic evidence of viral particles in tumor tissue (Kessler 1974; Nishiura 1983; Smith 1983). Yet, extensive epidemiologic, histologic, molecular, and microviral data now demonstrate that human papilloma virus (HPV) is the causative agent for cervical cancer; this knowledge is the basis for the current HPV vaccine (Vonsky 2019). The presence of talc in tissue removed from an individual ovarian cancer patient cannot be accepted as evidence of causality *per se*. Moreover, given the vast array of cancers that can occur in the ovary, it is inconceivable that any single endogenous or exogenous factor such as talc can be attributed to their diverse etiologies.

The presence of endometriosis in tissue adjacent to Tamara Newsome's low-grade endometrioid ovarian cancer, as well as foci of atypical endometriosis that merge with the carcinoma, the additional presence of mismatch repair protein deficiency in the tumor cells and uterine adenomyosis, as well as her relatively young age at diagnosis (53 years) provide compelling

evidence that this ovarian cancer arose from endometriosis. As stated earlier in the general section of my report, endometriosis is a well-recognized precursor of ovarian endometrioid carcinoma.

Figures

Figure 1. Low-grade endometrioid adenocarcinoma (FIGO grade 1) involving right ovary (100x).

Figure 2. Low-grade endometrioid adenocarcinoma (FIGO grade 1) involving uterine serosa (100x).

Figure 3. Low-grade endometrioid adenocarcinoma (top) arising in right ovary associated with endometriosis (bottom left) (20x).

Figure 4. Higher magnification of endometriotic gland depicted in Figure 3 (200x).

Figure 5. Atypical endometriosis (bottom left) associated with low-grade endometrioid adenocarcinoma (top right) in right ovary (100x).

Figure 6. Higher magnification of atypical endometriosis depicted in Figure 5. The glands are complex and the epithelium is atypical (200x).

Figure 7. Another focus of atypical endometriosis exhibits cytologic atypia only (200x).

Figure 8. The adenocarcinoma exhibits strong nuclear expression for PAX-8, which is a marker for mullerian (not colorectal) differentiation (200x).

Figure 9. The adenocarcinoma also exhibits strong expression for CK7, which is also typical for mullerian differentiation (200x).

Figure 10. The adenocarcinoma is negative for CK20, which is also typical for mullerian differentiation (200x). CK20 is typically positive in colorectal adenocarcinoma.

Figure 11. CD10 highlights the stroma around the endometriotic gland depicted in Figure 4 (200x).

Figure 12. Extensive adenomyosis is present in the uterus (20x).

Newsome v. Johnson & Johnson

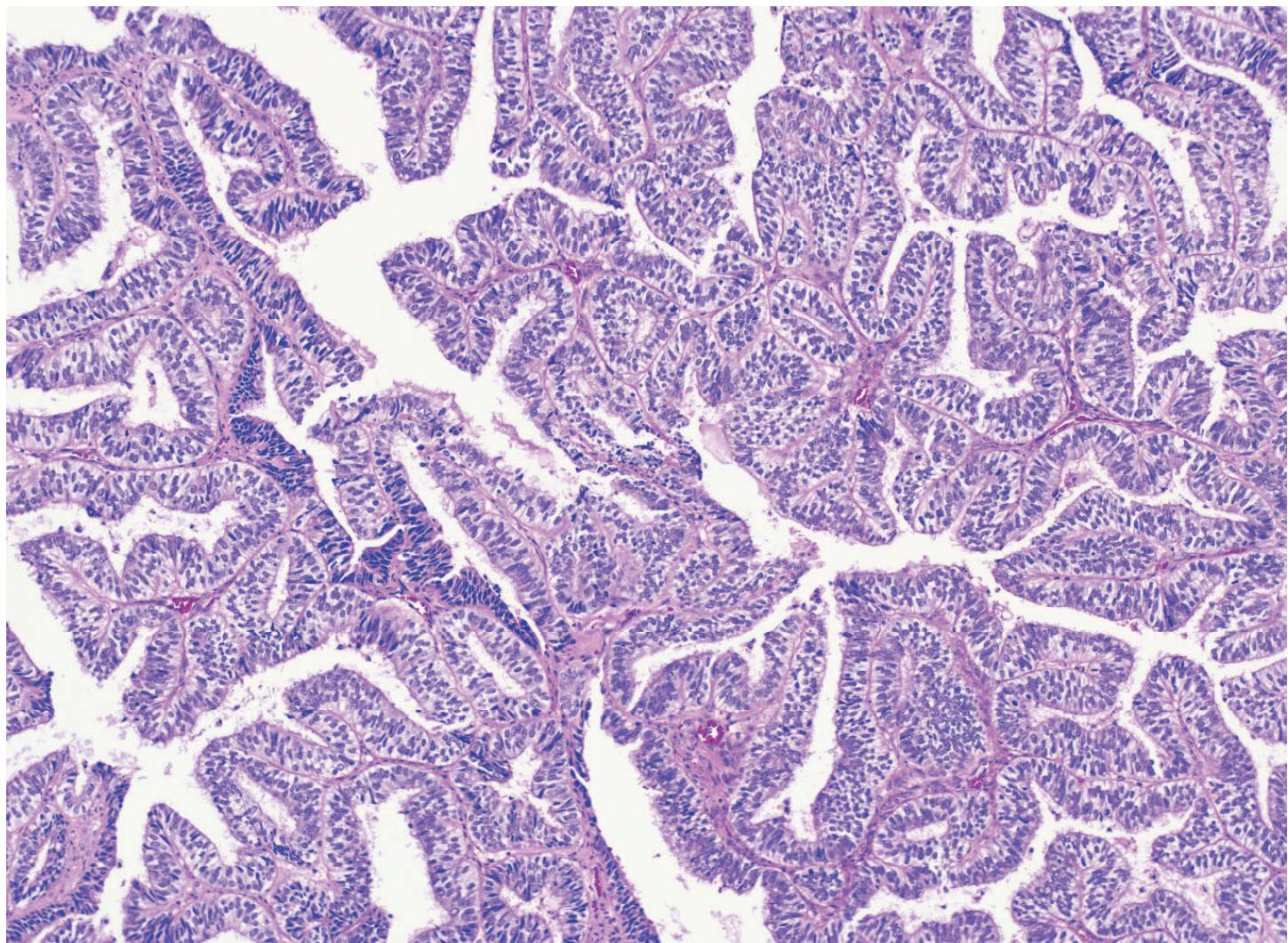


Figure 1. Low-grade endometrioid adenocarcinoma (FIGO grade 1) involving right ovary (100x).

Newsome v. Johnson & Johnson

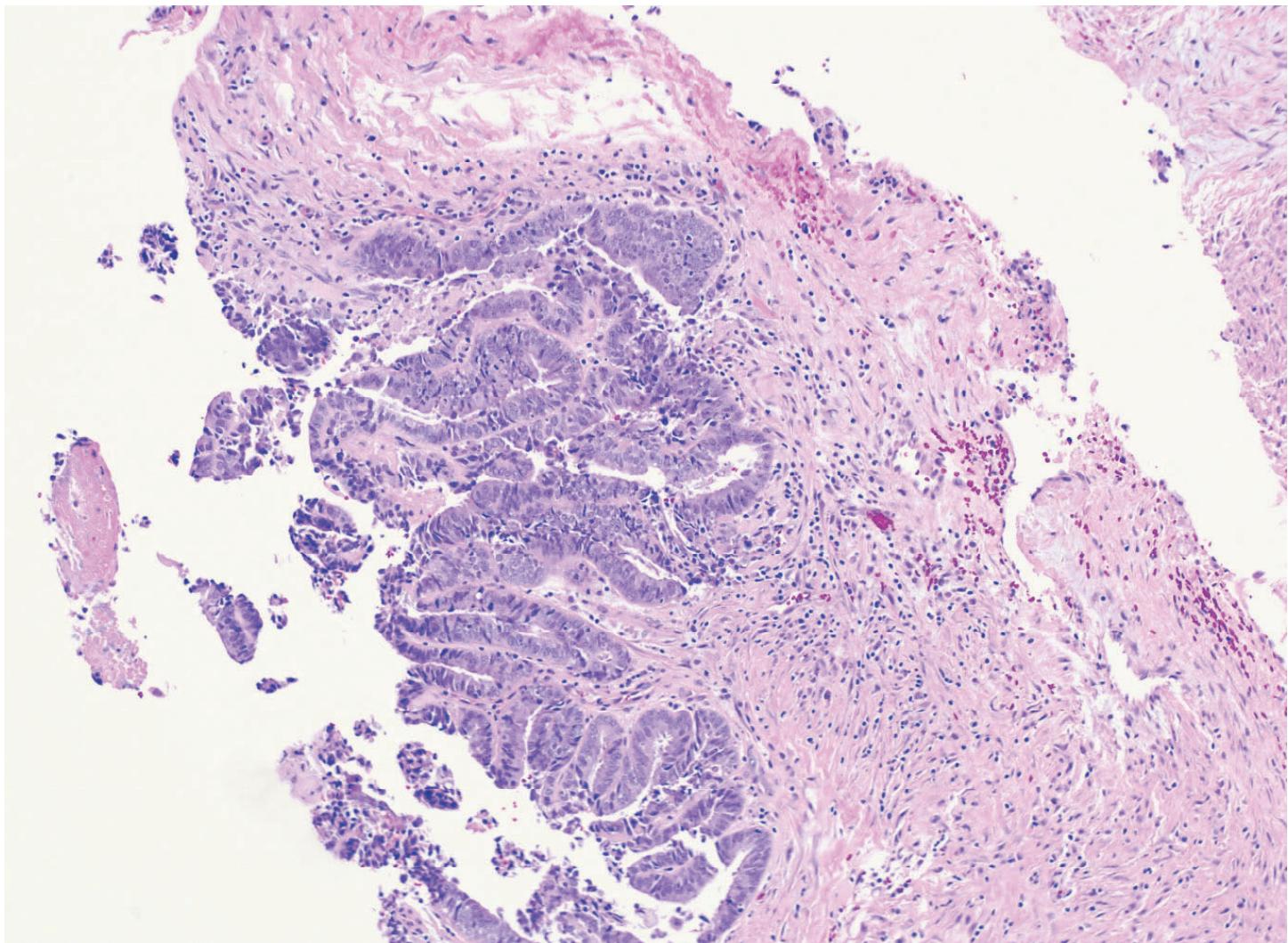


Figure 2. Low-grade endometrioid adenocarcinoma (FIGO grade 1) involving uterine serosa (100x).

Newsome v. Johnson & Johnson

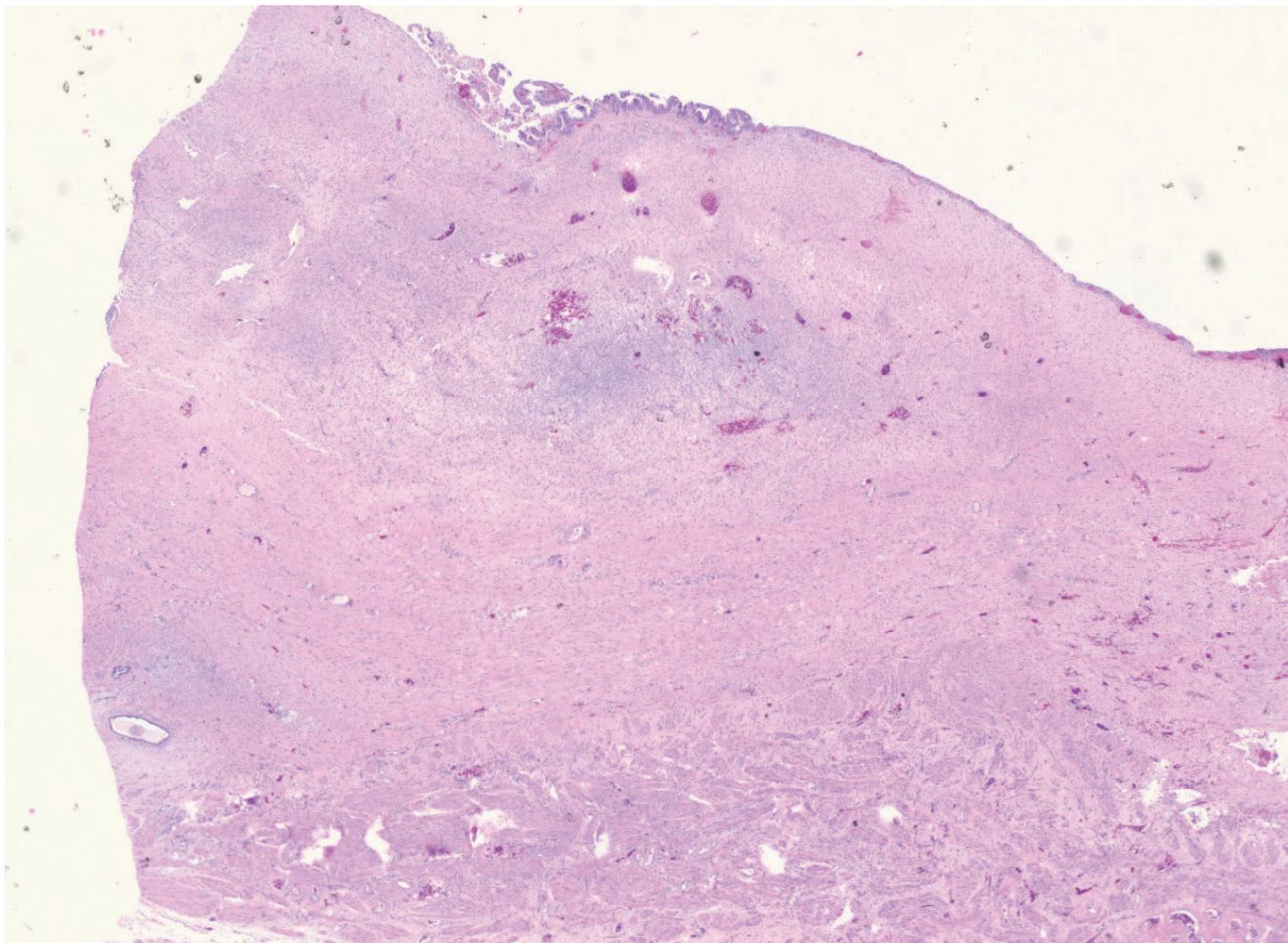


Figure 3. Low-grade endometrioid adenocarcinoma (top) arising in right ovary associated with endometriosis (bottom left) (20x).

Newsome v. Johnson & Johnson

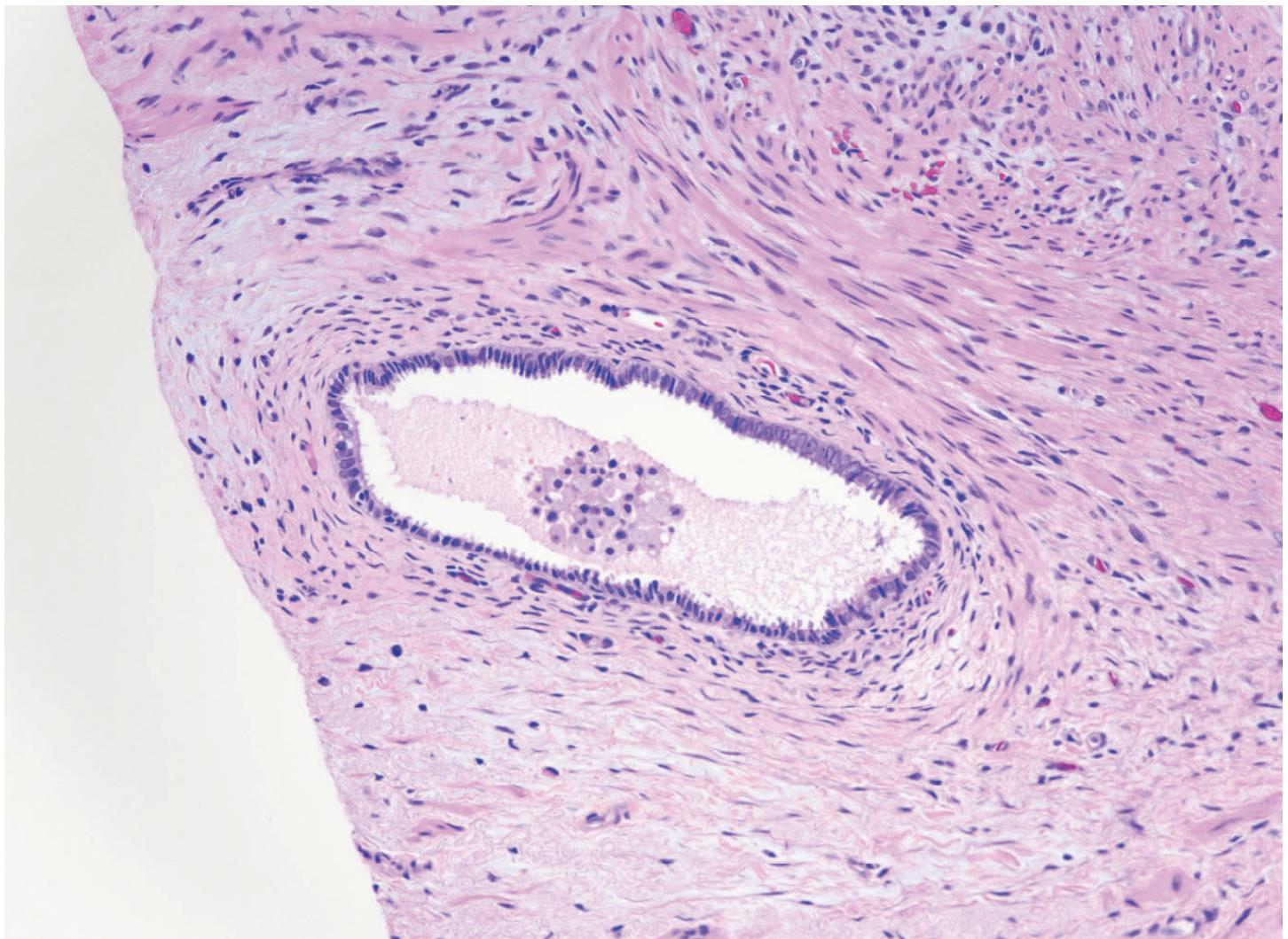


Figure 4. Higher magnification of endometriotic gland depicted in Figure 3 (200x).

Newsome v. Johnson & Johnson

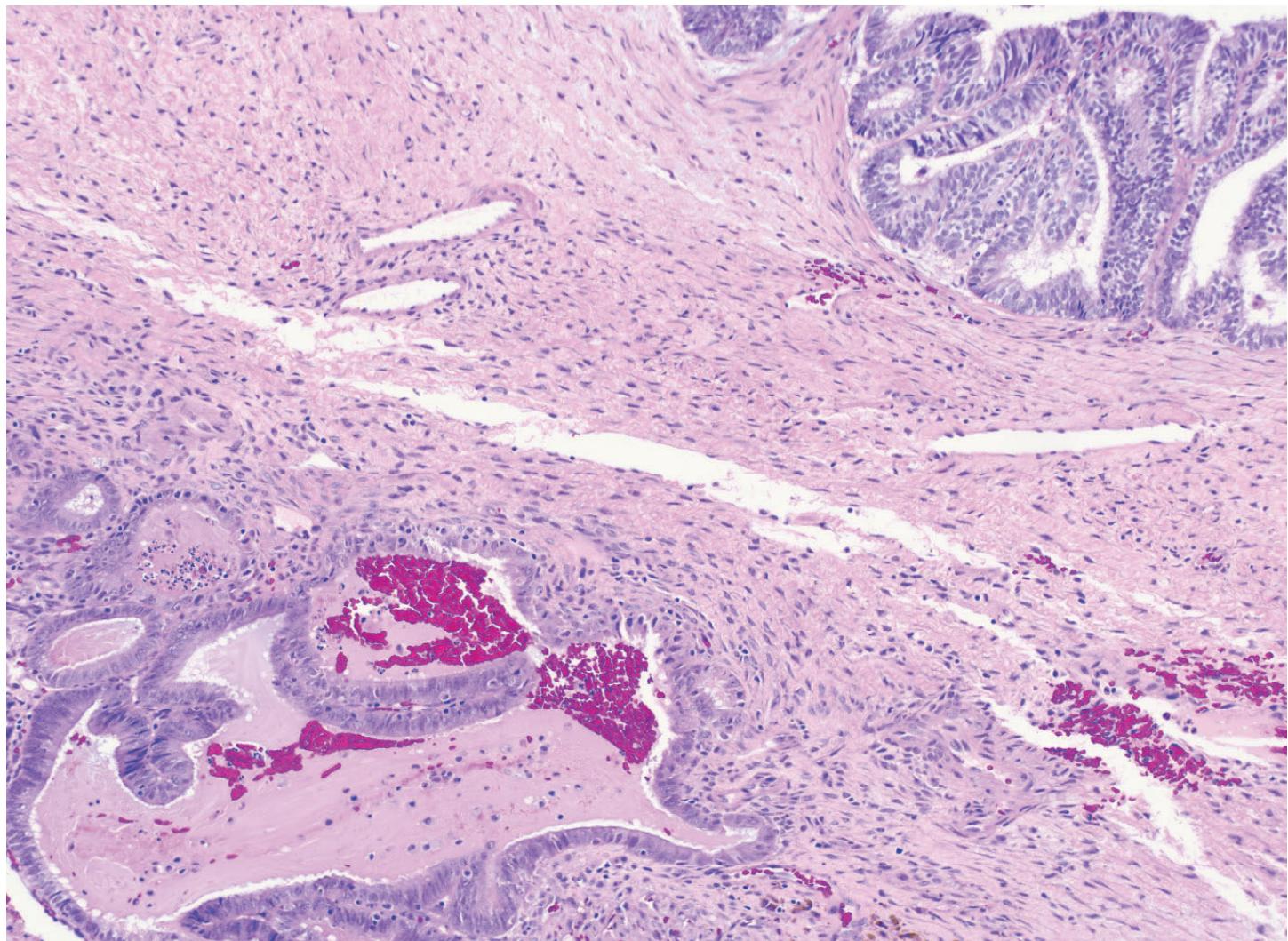


Figure 5. Atypical endometriosis (bottom left) associated with low-grade endometrioid adenocarcinoma (top right) in right ovary (100x).

Newsome v. Johnson & Johnson

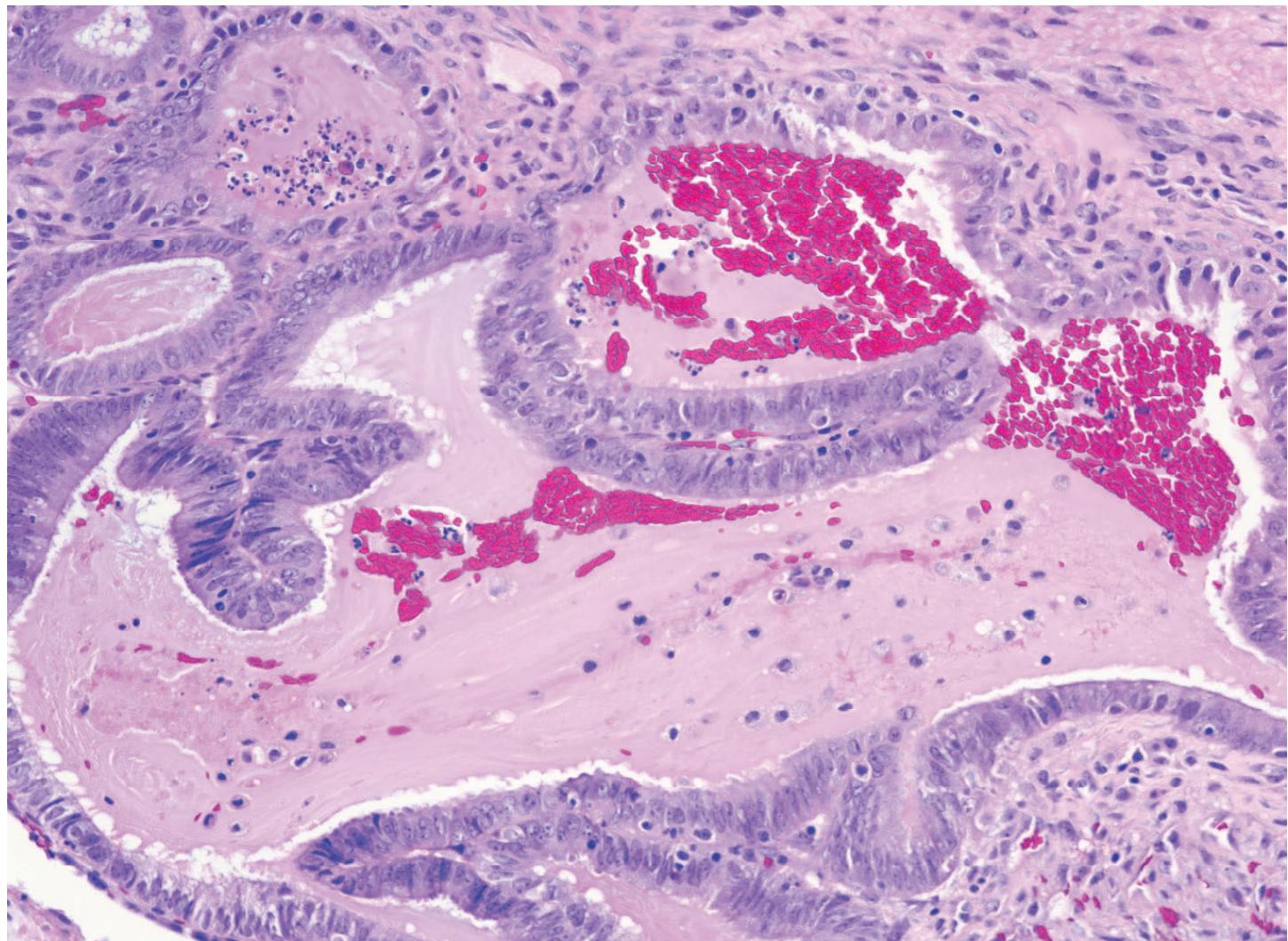


Figure 6. Higher magnification of atypical endometriosis depicted in Figure 5. The glands are complex and the epithelium is atypical (200x).

Newsome v. Johnson & Johnson

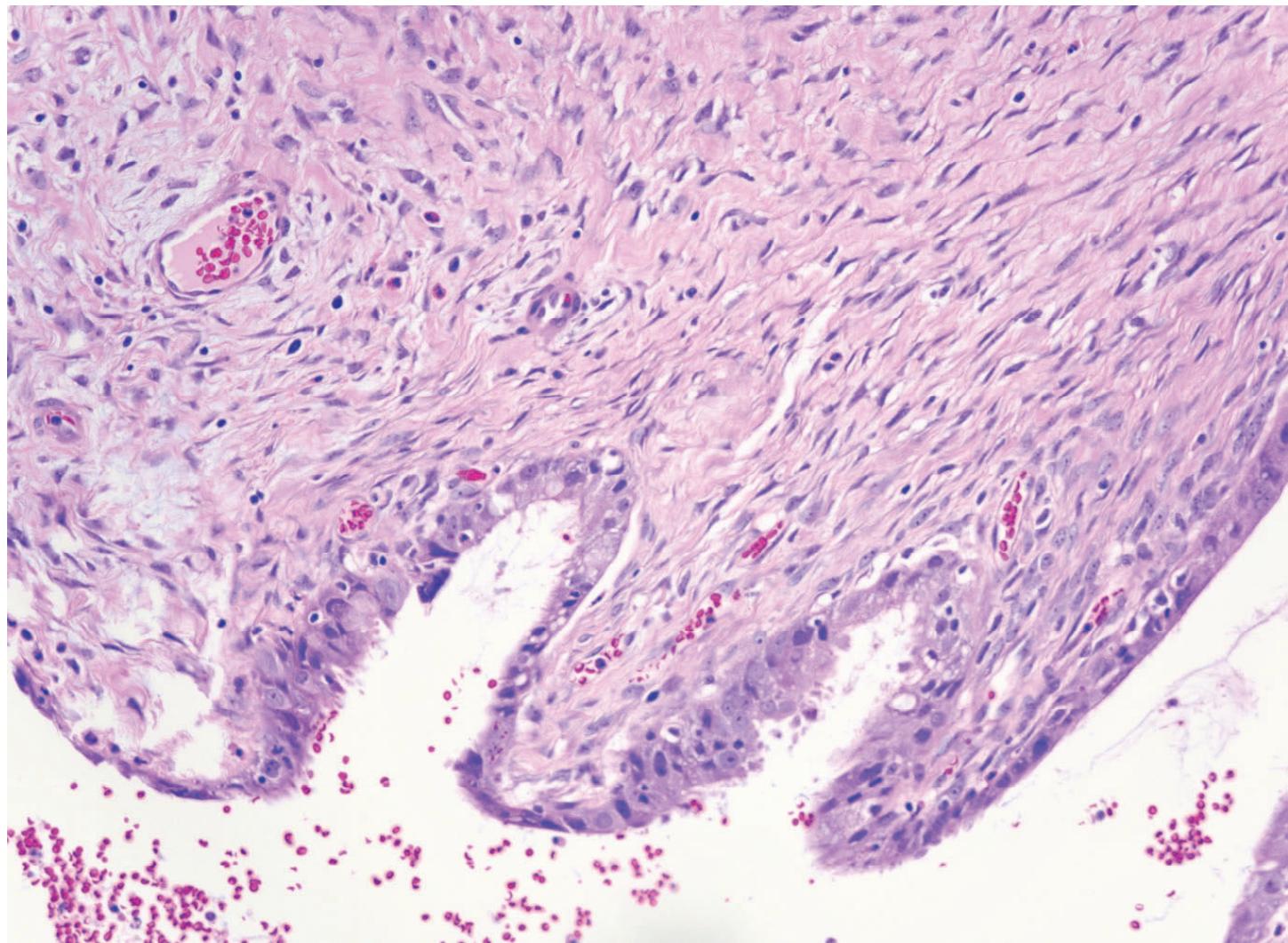


Figure 7. Another focus of atypical endometriosis exhibits cytologic atypia only (200x).

Newsome v. Johnson & Johnson

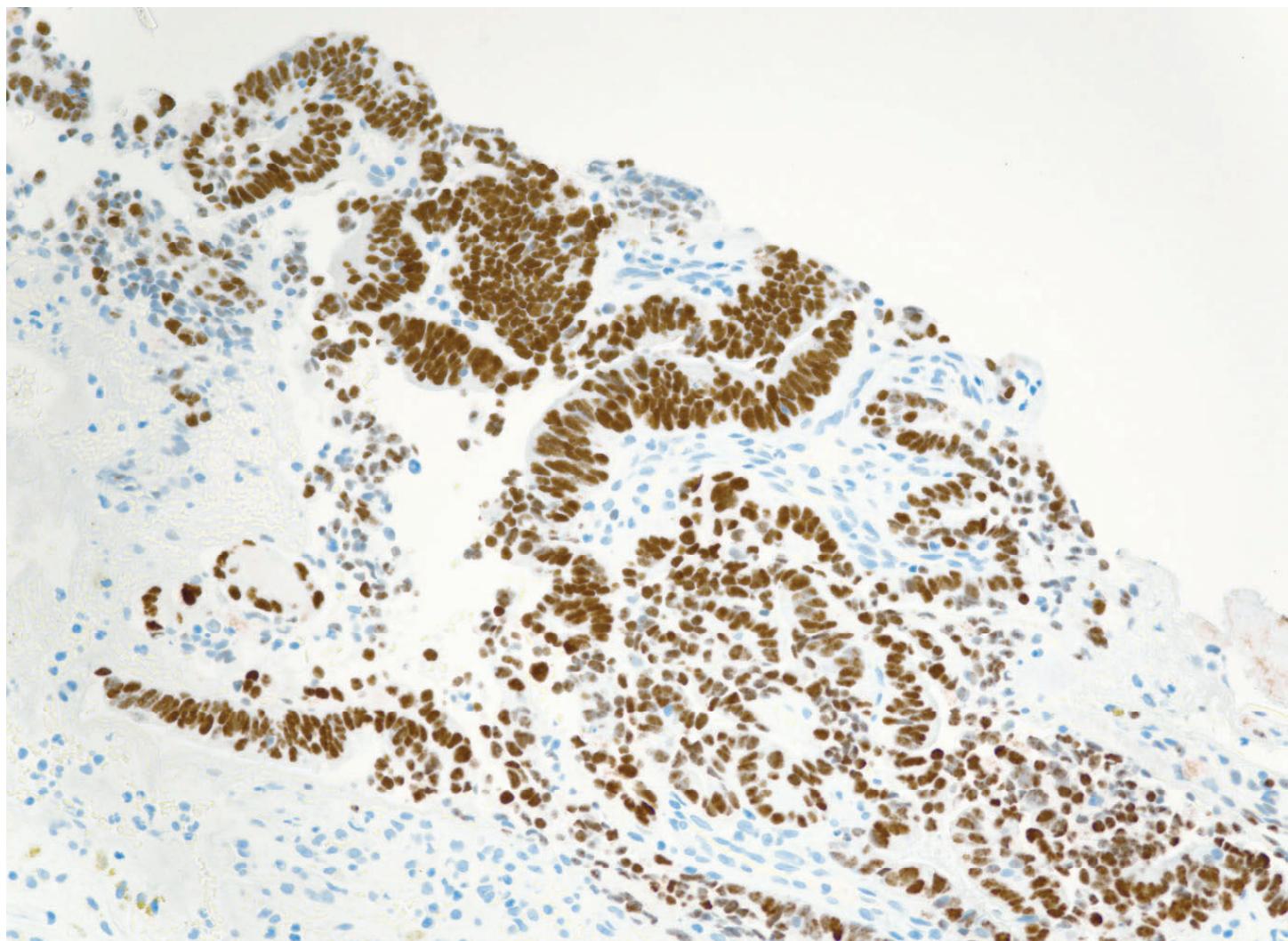


Figure 8. The adenocarcinoma exhibits strong nuclear expression for PAX-8, which is a marker for mullerian (not colorectal) differentiation (200x).

Newsome v. Johnson & Johnson

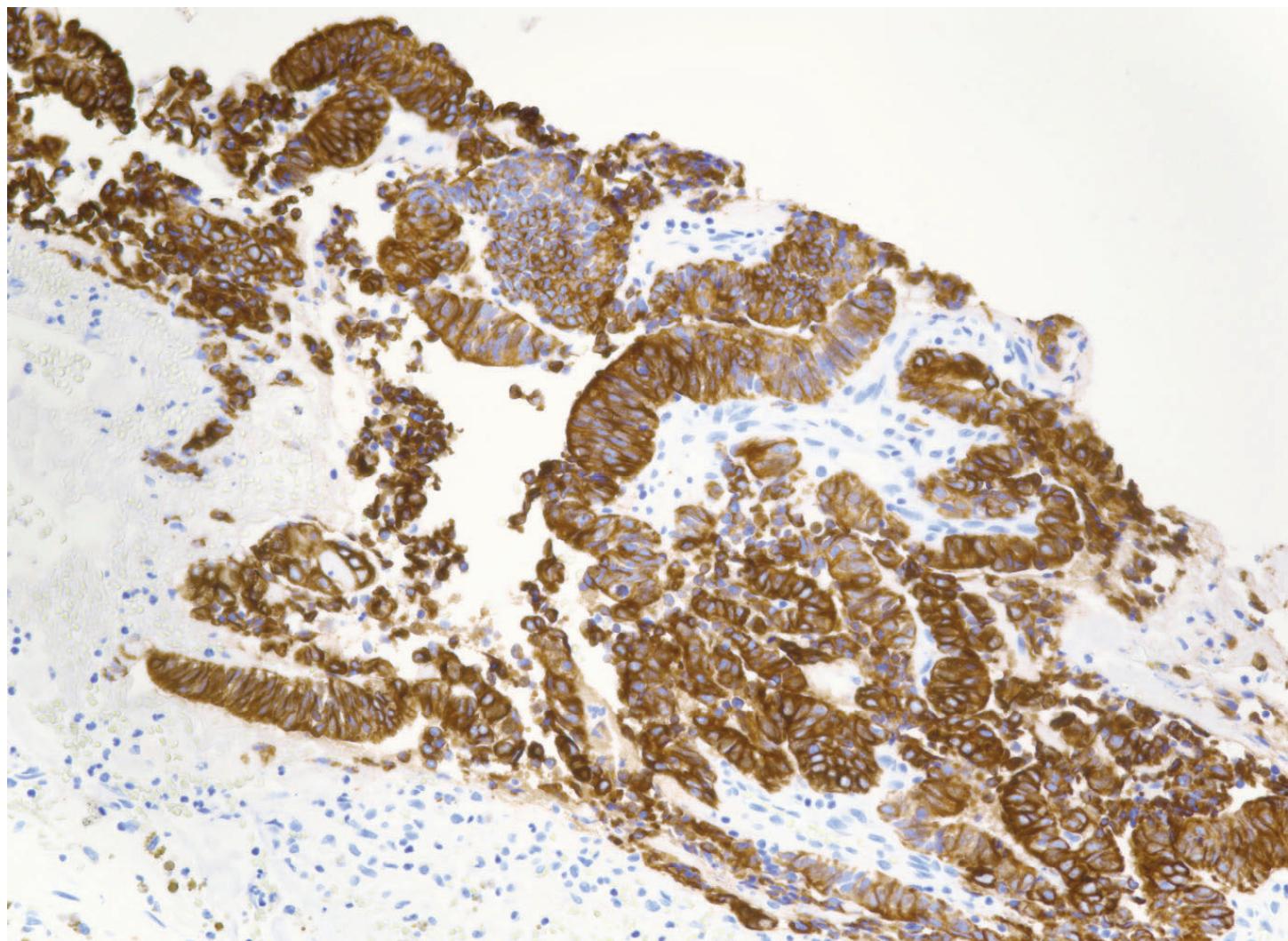


Figure 9. The adenocarcinoma also exhibits strong expression for CK7, which is also typical for mullerian differentiation (200x).

Newsome v. Johnson & Johnson

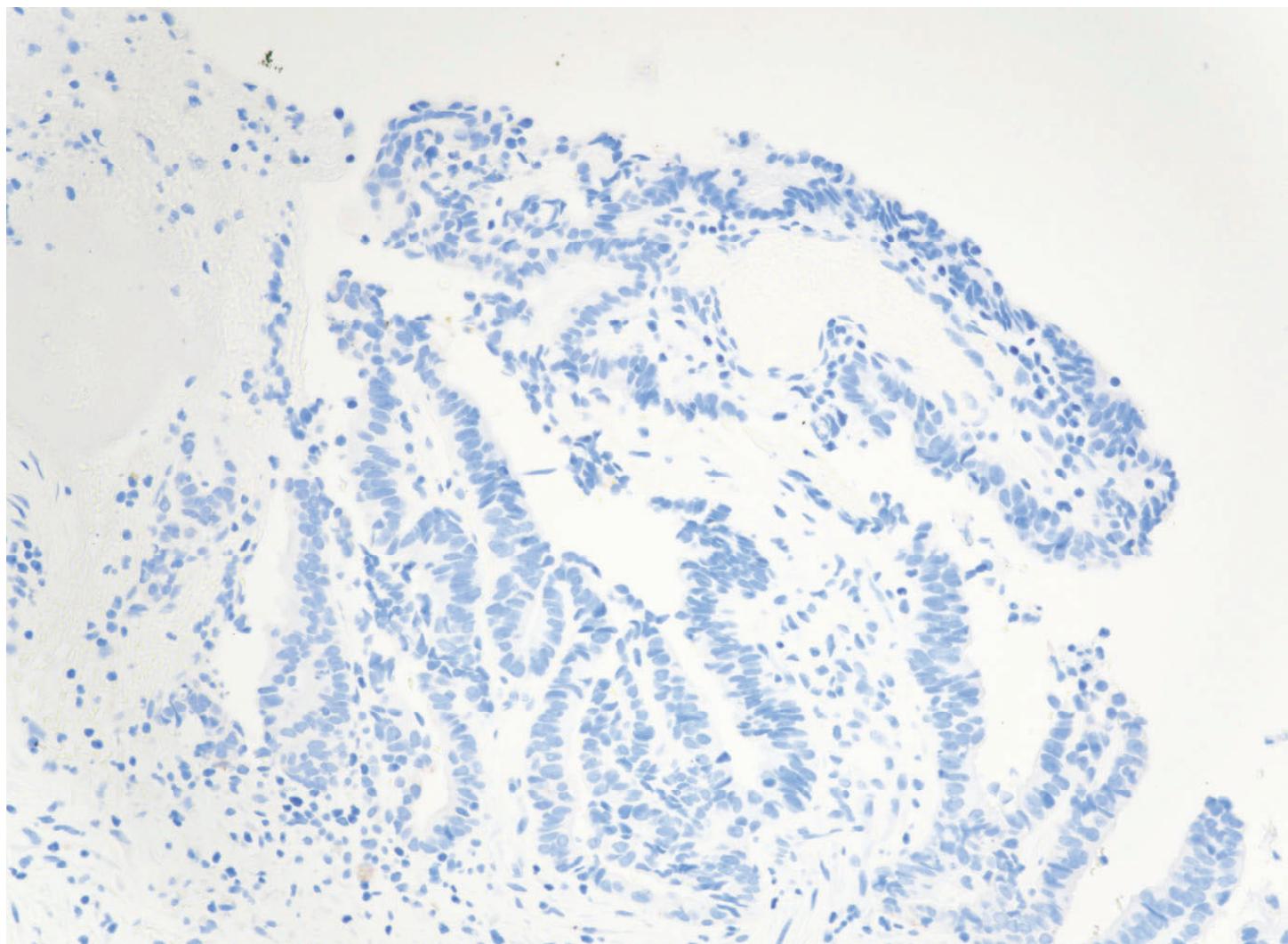


Figure 10. The adenocarcinoma is negative for CK20, which is also typical for mullerian differentiation (200x). CK20 is typically positive in colorectal adenocarcinoma.

Newsome v. Johnson & Johnson

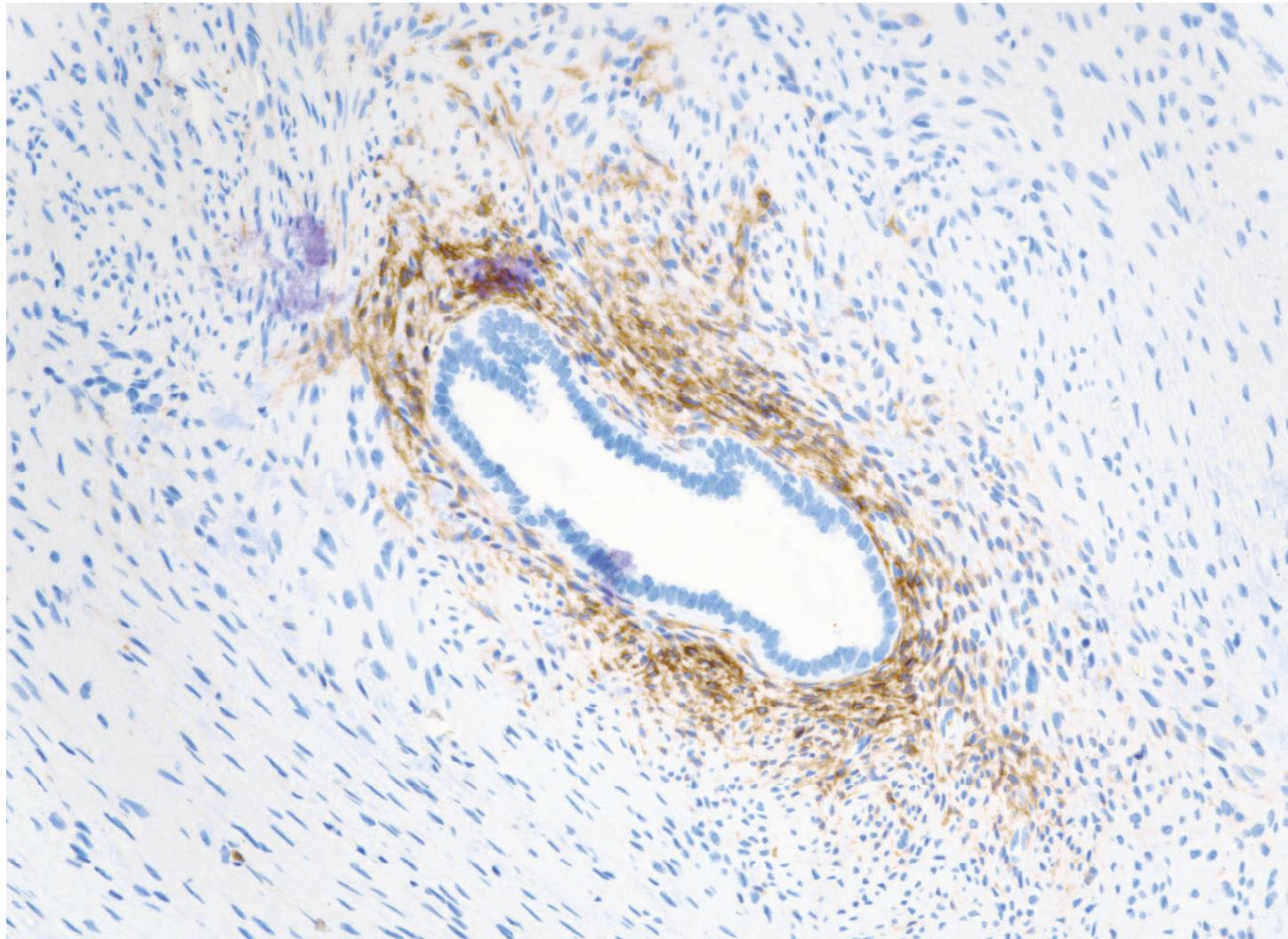


Figure 11. CD10 highlights the stroma around the endometriotic gland depicted in Figure 4 (200x).

Newsome v. Johnson & Johnson

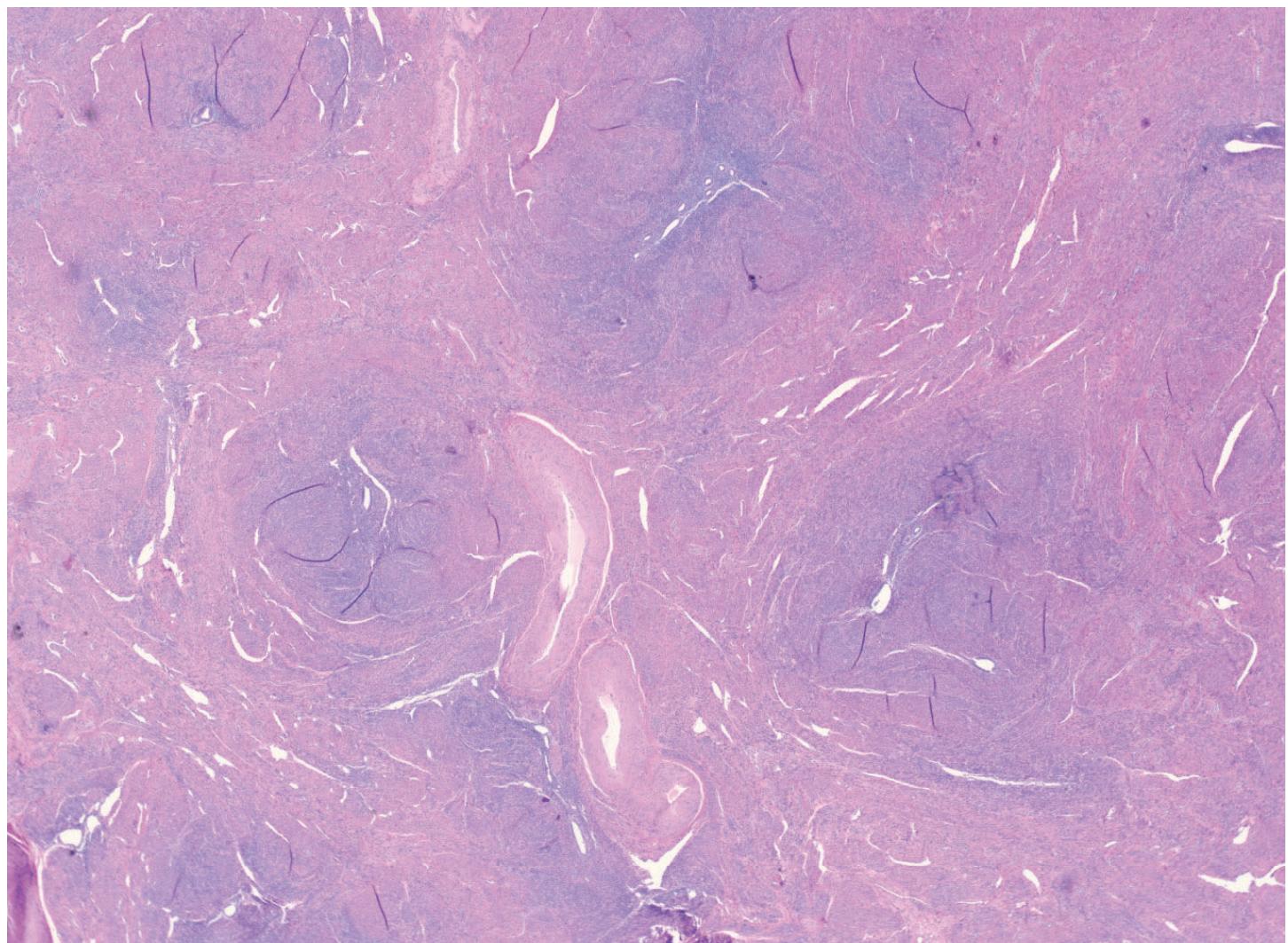


Figure 12. Extensive adenomyosis is present in the uterus (20x).

EXHIBIT A

CURRICULUM VITAE

PERSONAL DATA

Name: **Teri A. Longacre, M.D.**
Place of Birth: Los Angeles, California
Citizenship: U.S.A.
Spouse: Richard H. Hildebrandt, M.D.
Children: Nicole T. Hildebrandt, Jesse I. Hildebrandt, Daniella E. Hildebrandt
E-mail: longacre@stanford.edu
Telephone: 650-498-6460

EDUCATIONAL BACKGROUND

1972-1976 St. John's College, Santa Fe, New Mexico, B.A., Liberal Arts
1976-1980 University of New Mexico, Albuquerque, New Mexico, B.S., Biology
1981-1985 University of New Mexico School of Medicine, Albuquerque, New Mexico, M.D. (Degree awarded in December 1985)

POSTGRADUATE EDUCATION

1980-1981 Research Assistant (S.A. Bartow, M.D.), University of New Mexico School of Medicine, Albuquerque, New Mexico
1983-1984 Post-Sophomore Fellowship in Pathology, University of New Mexico School of Medicine, Albuquerque, New Mexico
1986-1987 Resident in Pathology, University of New Mexico Hospital, Albuquerque, New Mexico
1987-1988 Fellow in Hematopathology, University of New Mexico Hospital, Albuquerque, New Mexico
1988-1989 Fellow in Gastrointestinal Pathology, University of New Mexico Hospital, Albuquerque, New Mexico
1989-1990 Resident in Pathology, University of New Mexico Hospital, Albuquerque, New Mexico
1990-1991 Fellow in Surgical Pathology, Stanford University Hospital, Stanford, California
2004 Leadership Development for Physicians in Academic Health Centers, Harvard School of Public Health, Boston, Massachusetts
2005-2006 Stanford Physician Leadership Development Program, Stanford University School of Medicine, Stanford, California
2010-2011 Breast Prognostic Factors Testing, College of American Pathology, Chicago, Illinois
2017 Relationship-Centered Communication Skills Training Program, Stanford Health Care Communication Program & American Academy on Communication in Healthcare (AACH)

2018 Coach, Advancing Communication Excellence at Stanford (ACES)

ACADEMIC APPOINTMENTS

1991-1993	Clinical Instructor and Staff Physician, Department of Pathology, Stanford University Medical Center, Stanford, California
1993-1999	Assistant Professor of Pathology, Department of Pathology, Stanford University Medical Center, Stanford, California
1999-	Associate Professor of Pathology, Department of Pathology, Stanford University Medical Center, Stanford, California
2008-	Professor, Department of Pathology, Stanford University Medical Center Stanford, California

LICENSURE

1989	New Mexico, #89-123 (currently inactive)
1990	California, HG-069115
2011	Nevada, 14213

NATIONAL PROVIDER IDENTIFIER

2024	1528120896
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BOARD CERTIFICATION

1989	Diplomate, American Board of Medical Examiners
1991	Diplomate, American Board of Pathology, Anatomic and Clinical Pathology
2014	Voluntary Recertification, American Board of Pathology, Anatomic and Clinical Pathology

PROFESSIONAL MEMBERSHIPS

1990-	U.S. and Canadian International Academy of Pathologists
1996-	International Society of Gynecological Pathologists
1996-	South Bay Pathology Society
1997-	American Society of Clinical Pathologists
1997	International Society of Breast Pathology
1999	Gynecologic Oncology Group
2004	American Society of Clinical Oncology
2005-	College of American Pathologists
2006-	California Society of Pathologists
2007-	International Gynecologic Cancer Society
2008-	Arthur Purdy Stout Society
2010-	Association of Directors of Anatomic Surgical Pathology
2013-	The Rodger C. Haggitt Gastrointestinal Pathology Society
2016-	Pancreatobiliary Pathology Society

ADMINISTRATIVE AND SCIENTIFIC COMMITTEE APPOINTMENTS

1993-1994	Abstract Review Board, Gastrointestinal Pathology, United States and Canadian Academy of Pathologists
1994-2009	Admissions Panel, Stanford University School of Medicine, Stanford, California
1996-2008	Pathology Working Group Committee, National Cancer Institute, Breast and Ovarian Cancer Family Registry
2000-2001	Strategic Planning Committee on Research Space, Department of Pathology, Stanford University School of Medicine, Stanford, California
2000-2003	Alternate Senator, Faculty Senate, Stanford University School of Medicine, Stanford, California
2001-2009	Committee on Admissions, Stanford University School of Medicine, Stanford, California
2001-2002	Chair, Hematopathology Search Committee, Department of Pathology, Stanford University, Stanford, California
2001-2002	Committee on Women in Medicine and Science, Stanford University School of Medicine, Stanford, California
2001-2010	Co-Chair, Residency Selection Committee, Department of Pathology, Stanford University, Stanford, California
2002-2003	Renal Pathology Search Committee, Department of Pathology, Stanford University, Stanford, California
2001-	Adjunct Clinical Faculty Appointments and Promotions Committee, Department of Pathology, Stanford University, Stanford, California
2002-2004	Course Director, Surgical Pathology Clerkship 302A, Stanford University School of Medicine, Stanford, California
2002-2010	Associate Director, Residency Training Program, Department of Pathology, Stanford University, Stanford, California
2003-2008	Chair, Pediatric Pathology Search Committee, Department of Pathology, Stanford University, Stanford, California
2002-2009	Course Co-Director, Current Issues in Anatomic Pathology, University of San Francisco-Stanford Postgraduate Course, San Francisco, California
2004-2011	Associate Director of Surgical Pathology, Department of Pathology, Stanford University, Stanford, California
2004-2005	Co-Director, Women's Health Module, Human Health & Disease 223, Stanford University School of Medicine
2002-2003	Longitudinal Committee on Medical Education, Subcommittee on Admissions, Stanford University School of Medicine, Stanford, California
2005-2011	Cancer Care Committee, Stanford Comprehensive Cancer Center, Stanford, California
2005-	American Board of Pathology Test Development and Advisory Committee, The American Board of Pathology, Tampa, Florida
2005-2010	Associate Chair of Pathology for Residency Training, Department of Pathology, Stanford University, Stanford, California
2006-2007	Gynecologic Oncology Search Committee, Department of Gynecology,

2006-	Stanford University, Stanford, California Associate Member, Stanford Comprehensive Cancer Center, Stanford, California
2006-2012	Breast Oncology Program Director Search Committee, Stanford Comprehensive Cancer Center, Stanford, California
2007-	Gynecological Cancer Protocol Review Panel, College of American Pathologists
2007-	Education Committee, California Society of Pathologists
2007-2016	Director, Gynecologic and Breast Pathology Fellowship
2007-	Director, Gynecologic Pathology
2007-2011	Neuropathology Search Committee, Department of Pathology, Stanford University, Stanford, California
2007-2014	Treasurer, International Society of Gynecological Pathologists
2007-2013	Institutional Review Board, Stanford University School of Medicine, Stanford, California
2007-2008	Task Force on Industry Support of CME, Stanford University School of Medicine, Stanford, California
2008-2016	Chair, Quality Improvement Committee (PPEC), Department of Pathology, Stanford University School of Medicine, Stanford, California
2008-2012	National Cancer Institute, Breast Cancer Family Registry, Biospecimen Working Group
2009-2017	Medical Director, Stanford Medicine Outpatient Center Laboratory of Surgical Pathology, Stanford Medicine, Redwood City, California
2009-	AANCART California Biorepository Research Network
2009-2014	Arthur Purdy Stout Society Prize and Awards Committee, Arthur Purdy Stout Society
2010-	Council, Association of Directors of Anatomic Surgical Pathology
2010-2012	Co-Director of Surgical Pathology, Department of Pathology, Stanford University, Stanford, California
2011-	Ambassador, United States and Canadian Academy of Pathology
2011-2014	Tissue Committee, Lucile Packard Children's Hospital, Stanford, California
2011-2016	Care Improvement Committee, Stanford University Hospital, Stanford, California
2011-	Director, Gastrointestinal Pathology
2011-2012	Physicianship and Leadership Working Group, Transforming Medical Education Initiative, Stanford School of Medicine, Stanford, California
2012	CAP/ASCCP Lower Anogenital Squamous Terminology (LAST) Consensus Statement Independent Review Panel
2012-2018	Director, Stanford Tissue Procurement Facility, Stanford Cancer Center
2012	Gynecologic Pathology Search Committee, Department of Pathology, Stanford University, Stanford, California
2012-2017	Senator-at-Large, Stanford Medical School Faculty Senate, Stanford University, Stanford, California
2012	Councilor, International Academy of Pathology, US-Canadian Division

2013	Molecular Therapeutics in Gynecologic Oncology Search Committee, Division of Gynecologic Oncology, Department of Obstetrics and Gynecology and Division of Radiation & Cancer Biology, Department of Radiation Oncology, Stanford, California
2013	World Health Organization Tumors of the Female Genital Tract Consensus Meeting, Lyon, France
2013-2018	Endometrial Cancer Biomarker Reporting Panel, College of American Pathologists
2013-	American Medical Foundation for Peer Review and Education
2013-2017	Delegate to the California Delegation, College of American Pathologists House of Delegates
2013-	Scientific Advisory Board, Stop GCT, Ovarian Cancer Research Foundation
2013-	Director, Gastrointestinal Pathology Fellowship
2014-	Consultant, American Medical Foundation for Peer Review & Education
2014-	Vice President, Association of Directors of Anatomic and Surgical Pathology
2014-2016	Education Committee, International Society of Gynecological Pathologists
2014-	Cancer Biomarker Reporting Committee, College of American Pathologists
2014-	Molecular Oncology Tumor Board, ASCO University and College of American Pathologists
2014-2016	Chair, Stanford Hospital Tissue Committee, Stanford Health Care, Stanford, California
2014-	Action Group: Pathology Practice Guidance, College of American Pathologists
2014-2017	Chair, Endometrial Cancer Biomarker Reporting Panel, College of American Pathologists
2014	Faculty Search Committee, Department of Pathology, Stanford, California
2014	Councilor, International Academy of Pathology, US-Canadian Division
2016-2018	President, Association of Directors of Anatomic and Surgical Pathology
2016-	Steering Committee, Gynecologic Cancer InterGroup (GCIG) Gynecologic Oncology Pathology Working Group
2016	Organizer, Scientific Symposiums International, Annual Dr. Richard L. Kempson Surgical Pathology Course
2016-	Director, Gynecologic Pathology Fellowship
2017	NIDDK Liver Tissue and Cell Distribution System Review Panel
2017	US Pathology Biomarker Advisory Board, Merck
2017	Well-Being Directors Council, Stanford WellMD Center, Stanford, California
2018	NCI Specialized Programs of Research Excellence (SPORE) Review Panel
2018	NCI Special Emphasis Panel, Feasibility and Planning Studies for

	Development of Specialized Programs of Research Excellence (SPOREs) to Investigate Cancer Health Disparities (P20)
2018	Advisory Panel, Editorial Board of Cancer.Net, American Society of Clinical Oncology
2018	Pathology Department Liaison for Wellness, Inclusion, and Diversity
2019	NCI Human Tumor Atlas Network (HTAN) Biospecimen Working Group
2019	World Health Organization Tumors of the Female Genital Tract Expert
	Editorial Board, International Agency for Research on Cancer
2019	American Society of Clinical Oncology Cancer.Net Review Panel, Ovarian, Fallopian Tube, and Peritoneal Cancer
2019	California Comprehensive Cancer Control Plan Colorectal Cancer Subcommittee
2019	California Comprehensive Cancer Control Plan Cervical Cancer / HPV Subcommittee
2019	Gastric Cancer Advisory Board, Astellas
2019	Oncology GI-GU Search Committee, Stanford Cancer Center
2019	Appointment and Promotion Oversight Committee, Department of Pathology, Stanford California
2019	United States & Canadian Academy of Pathology Mentoring Academy
2020	NCI Specialized Programs of Research Excellence (SPORE) Review Panel
2020	American Society of Clinical Oncology Cancer.Net Review Panel, Ovarian, Fallopian Tube, and Peritoneal Cancer
2020	European Society of Gynecological Oncology & Gynecologic Cancer Intergroup Borderline Ovarian Tumors Working Group
2020	Body Imaging Faculty Search Committee, Department of Radiology, Stanford California
2020	Member, Gynecologic Cancer Section, Faculty Opinions, F1000 Prime
2021	Member, Board of Directors, United States and Canadian Society of Pathology
2021	McCormick and Gabilan Faculty Award Review Committee, Office of Faculty Development and Diversity, Stanford School of Medicine
2022	Member, Gastroenterology Inflammatory Bowel Disease Faculty Search Committee, Division of Gastroenterology and Hepatology in the Department of Medicine
2022	Mersana Therapeutics Advisory Board
2023	AstraZeneca US Precision Medicine Pathologist Steering Committee
2024	Medical Advisory Board: GI Manifestations of Indolent Systemic Mastocytosis, Blueprint Medicines
2024	Verastem Pathology Advisory Board

EDITORIAL BOARD

1993-	Advances in Anatomic Pathology
1993-2001	Advances in Gastroenterology, Hepatology and Clinical Nutrition
1996-	International Journal of Gynecological Pathology

2003-	Applied Immunohistochemistry and Molecular Morphology
2005-	American Journal of Surgical Pathology
2009-	Associate Editor, Digestive Diseases and Sciences, Stanford Multidisciplinary Seminars
2009-	Pathology Research International
2009-2020	Modern Pathology
2009-	PathXchange Editorial Panel
2013-	Pathology Discovery (Senior Editor)
2013-	Seminars in Diagnostic Pathology
2014-	PLoS ONE
2016	Journal of Gastroenterology, Hepatology and Endoscopy
2016-	American Journal of Surgical Pathology: Reviews & Report
2021-	European Journal of Gynaecologic Oncology

Journal Ad Hoc Reviewer:

American Journal of Gastroenterology
American Journal of Obstetrics and Gynecology
American Journal of Pathology
Annals of Oncology
Archives of Pathology and Laboratory Medicine
BMC Cancer
BMC Gastroenterology
British Journal of Cancer
Cancer
Cancer Epidemiology, Biomarkers and Prevention
Cancer Research
Clinical Medicine-Pathology
Clinical Microbiology and Infection
Colorectal Disease
Digestive Diseases and Sciences
Expert Review of Anticancer Therapy
Gastrointestinal Cancer: Targets and Therapy
Gut
Gynecologic Oncology
Histology and Histopathology
Histopathology
Human Pathology
International Journal of Gynecological Cancer
International Journal of Medical Sciences
Journal of Clinical Oncology
Journal of Obstetrics and Gynaecology
Journal of Pathology
Journal of Reproductive Medicine
Journal of Zhejiang University-SCIENCE B
Medical Science Monitor

Molecular and Cellular Biology
Molecular Cancer Therapeutics
Obstetrics and Gynecology International
Oncogene
The Lancet
The Lancet Digital Health
The Surgery Journal
Virchows Archives
World Journal of Surgical Oncology

External Grant Reviewer:

American Institute of Biological Sciences
Dutch Cancer Society
French National Cancer Institute
Irish Health Research Board
Italian Association for Cancer Research
Physicians' Services Incorporated Foundation
Qatar National Research Fund

HONORS AND AWARDS

1984	Khatali Award in Recognition of an Outstanding Medical Student, University of New Mexico School of Medicine, Albuquerque, New Mexico
1984	Faculty Award of Excellence, University of New Mexico School of Medicine, Albuquerque, New Mexico
1984	Gordon Award in Pathology, University of New Mexico School of Medicine, Albuquerque, New Mexico
1994	Katharine McCormick Faculty Award, Stanford University Medical Center
1996	American Cancer Society Clinical Oncology Career Development Award
1996	American Cancer Society Institutional Research Grant
2003	Service Award for Academic Advising, Undergraduate Advising Center, Stanford University, Stanford, California
2010	Excellence in Teaching, Women's Health Unit, Human Health and Disease, Stanford University School of Medicine, Stanford, California
2012	Excellence in Teaching, Women's Health Unit, Human Health and Disease, Stanford University School of Medicine, Stanford, California
2016	Excellence in Teaching Nominee, Human Health and Disease, Stanford University School of Medicine, Stanford, California
2018	ACES Honorary Award, Stanford Health Care, Stanford, California
2018	Richard L Kempson Endowed Professor of Surgical Pathology

TEACHING ACTIVITIES

1993-2005	Pathology 230C Lecturer: Endometrium, Myometrium & Fallopian Tube, Ovary I & II, Stanford University School of Medicine, Stanford, California
1993-2005	Pathology 230C Laboratory Instructor & Small Group Session Leader: Gynecologic & Breast Pathology, Stanford University School of Medicine, Stanford, California
1993-2005	Pathology 230C Laboratory Instructor and Small Group Session Leader: Endocrine and Bone Pathology, Stanford University School of Medicine, Stanford, California
1994	Lecturer, Anatomic Pathology Residency Training Program: GI Pathology I-VI, Department of Pathology, Stanford University, Stanford, California
1994-2005	Pathology 230C Lecturer: Breast Pathology I & II, Stanford University School of Medicine, Stanford, California
1995-2005	Lecturer, Anatomic Pathology Residency Training Program: Gyn Pathology, Department of Pathology, Stanford University, Stanford, California
1996	Adult GI Clinical-Pathologic Correlation Conference, Laboratory of Surgical Pathology, Stanford University Hospital and Medical Center, Stanford, California
2000-2005	Endocrine Pathology Laboratory Coordinator, Stanford University School of Medicine, Stanford, California
2003	Pediatric GI Clinical-Pathologic Correlation Conference, Laboratory of Surgical Pathology, Stanford University Hospital and Medical Center, Stanford, California
2004	Anatomic Pathology Didactic Lecture Series Organizer, Department of Pathology, Stanford University, Stanford, California
2005-2006	Human Health & Disease 223 Lecturer: Endometrium, Myometrium & Fallopian Tube, Ovary I & II, Stanford University School of Medicine, Stanford, California
2005-2006	Human Health & Disease 223 Lecturer: Breast I & II, Stanford University School of Medicine, Stanford, California
2005-2006	Human Health & Disease 223 Small Group Session Leader: Gynecologic Pathology, Stanford University School of Medicine, Stanford, California
2005-2006	Human Health & Disease 223 Small Group Session Leader: Breast Pathology, Stanford University School of Medicine, Stanford, California
2005	Lecturer, Anatomic Pathology Residency Training Program: Ovarian Neoplasia I-VI, Department of Pathology, Stanford University, Stanford, California
2008	Digestive Disease Conference, Stanford University School of Medicine, Stanford, California
2008	Human Health & Disease 223 Lecturer: GI Pathology I-II, Stanford University School of Medicine, Stanford, California
2008	Annual QA/QI Lecture: Quality in the Laboratory and Regulatory Agencies, Department of Pathology, Stanford University, Stanford, California
2008	Human Health & Disease 223 Lecturer: Endometrium, Myometrium &

	Fallopian Tube, Ovary I & II, Stanford University School of Medicine, Stanford, California
2008	Human Health & Disease 223 Small Group Session Leader: Gynecologic Pathology, Stanford University School of Medicine, Stanford, California
2009	Digestive Disease Conference, Stanford University School of Medicine, Stanford, California
2009	Human Health & Disease 223 Lecturer: GI Pathology I-II, Stanford University School of Medicine, Stanford, California
2009	Human Health & Disease 223 Lecturer: Endometrium, Myometrium & Fallopian Tube, Ovary I & II, Stanford University School of Medicine, Stanford, California
2010	Human Health & Disease 223 Lecturer: Myometrium & Fallopian Tube, Ovary I & II, Stanford University School of Medicine, Stanford, California
2011	Pediatric GI Pathology, Division of Pediatric Gastroenterology, Lucile Packard Children's Hospital, Palo Alto, California
2011	Human Health & Disease 223 Lecturer: Endometrium, Myometrium & Fallopian Tube, Ovary I & II, Stanford University School of Medicine, Stanford, California
2012	Human Health & Disease 223 Lecturer: GI Pathology I, Stanford University School of Medicine, Stanford, California
2012	Human Health & Disease 223 Lecturer: Endometrium, Myometrium & Fallopian Tube, Ovary I & II, Stanford University School of Medicine, Stanford, California
2012	Gastroenterology Fellows: Colorectal Pathology, Stanford University School of Medicine, Stanford, California
2013	Pediatric GI Pathology, Division of Pediatric Gastroenterology, Lucile Packard Children's Hospital, Palo Alto, California
2013	Gastroenterology Fellows: Colorectal Pathology, Stanford University School of Medicine, Stanford, California
2013	Human Health & Disease 223 Lecturer: GI Pathology I, Stanford University School of Medicine, Stanford, California
2013	Human Health & Disease 223 Small Group Session Leader: Liver Pathology, Stanford University School of Medicine, Stanford, California
2013	Human Health & Disease 223 Lecturer: Myometrium & Fallopian Tube, Ovary I & II, Stanford University School of Medicine, Stanford, California
2013	Human Health & Disease 223 Small Group Session Leader: Gynecologic Pathology, Stanford University School of Medicine, Stanford, California
2014	Gastroenterology Fellows: Pathology of the Upper Gastrointestinal Tract, Stanford University School of Medicine, Stanford, California
2014	Human Health & Disease 223 Lecturer: GI Pathology I, Stanford University School of Medicine, Stanford, California
2014	Human Health & Disease 223 Small Group Session Leader: Gastrointestinal Pathology, Stanford University School of Medicine, Stanford, California

2014	Human Health & Disease 223 Small Group Session Leader: Liver Pathology, Stanford University School of Medicine, Stanford, California
2014	Human Health & Disease 223 Co-Lecturer: Endometrium, Stanford University School of Medicine, Stanford, California
2014	Human Health & Disease 223 Lecturer: Myometrium & Fallopian Tube, Ovary I & II, Stanford University School of Medicine, Stanford, California
2014	Human Health & Disease 223 Small Group Session Leader: Gynecologic Pathology, Stanford University School of Medicine, Stanford, California
2015	Gastroenterology Fellows: Pathology of the Lower Gastrointestinal Tract, Stanford University School of Medicine, Stanford, California
2015	Human Health & Disease 223 Lecturer: Ovary, Stanford University School of Medicine, Stanford, California
2015	Human Health & Disease 223 Lecturer: Endometrium, Stanford University School of Medicine, Stanford, California
2015	Human Health & Disease 223 Lecturer: Lower GI Tract, Stanford University School of Medicine, Stanford, California
2015	Human Health & Disease 223 Small Group Session Leader: Gynecologic Pathology, Stanford University School of Medicine, Stanford, California
2016	Human Health & Disease 223 Lecturer: Lower GI Tract I & II, Stanford University School of Medicine, Stanford, California
2017	Human Health & Disease 223 Lecturer: Lower GI Tract I & II, Stanford University School of Medicine, Stanford, California
2018	Gastroenterology Fellows: Colorectal Polyps: A Managerial Approach, Stanford University School of Medicine, Stanford, California

POST-DOCTORAL FELLOWS (GYNECOLOGIC PATHOLOGY)

2007-2008	Ghada Esheba, MSc., M.D., Tanta Hospital, Tanta, Egypt
2008-2009	Amy Ly, M.D., Massachusetts General Hospital, Boston, Massachusetts
2009-2010	William Rogers, M.D., El Camino Hospital, Mountain View, California
2010-2011	Jonathan Kitayama, M.D., Kaiser Permanente, Honolulu, Hawaii
2011-2012	Saul Offman, M.D., Dalhousie University, Halifax, Nova Scotia, Canada
2012-2013	Lorraine Pan, M.D., AmeriPath, Denver, Colorado
2013-2014	Michael R. Clay, M.D., University of Colorado Medicine, Denver, Colorado
2013-2014	Christopher Conklin, M.D., Surrey Memorial Hospital, British of Columbia, Canada
2014-2015	Mary O'Keefe, M.D., Denver Health Medical Center, Denver, Colorado
2015-2016	Laura Moench, M.D., Fairview Southdale Hospital, Minneapolis, Minnesota
2015-2016	Sucheta Srivastava, M.D., Associated Pathology Medical Group, Los Gatos, California
2016-2017	Sydney Card, M.D., The University of British Columbia, Vancouver British of Columbia, Canada
2016-2017	Koah Vierrkoetter M.D., The Queen's Medical Center, Honolulu, Hawaii

2017-2018	Dina Kokh, M.D., Fraser Health Authority, Vancouver, British Columbia, Canada
2017-2018	Soufianne El Hallani, M.D., University of Alberta, Royal Alexandra Hospital - Women's Hospital, Edmonton, Canada
2017-2018	Eric Gars, M.D., Stanford University, Stanford, California
2018-2019	Keegan Barry-Holson, M.D., Kaiser Permanente South San Francisco, California
2018-2019	David Levy, M.D., John Muir Medical Center, Walnut Creek, California
2018-2019	Megan Fitzpatrick, M.D., University of Wisconsin, Madison, Wisconsin
2019-2020	Kelly Devereaux, M.D., Ph.D., New York University, New York City, New York
2019-2020	Juliana Weiel, M.D., Billings Clinic, Billings, Montana
2020-2021	Kevin Kohali, M.D., Stanford University, Stanford, California
2020-2021	Jenifer Pors, M.D., The University of British Columbia, Vancouver British of Columbia, Canada
2020-2021	Ellen Cai, M.D., Kelowna General Hospital, Kelowna, British Columbia
2021-2022	Erna Forgo, Cleveland Clinic, Cleveland, Ohio
2021-2022	Lucy Han, M.D., California Pacific Medical Center, San Francisco, California
2021-2022	Emily Ryan, M.D. Stanford University, Stanford, California
2022-2023	Xiaoming Zhang, MD, Stanford University, Stanford California
2022-2023	Troy Tenney, MD, University of New Mexico, Albuquerque, New Mexico
2023-2024	Ashley Monsrud, MD, Stanford University, Stanford California
2023-2024	Austin McHenry, MD, Stanford University, Stanford California

POST-DOCTORAL FELLOWS (GASTROINTESTINAL PATHOLOGY)

2013-2014	Michael DiMaio, M.D., Companion Diagnostics, Carpinteria, California
2014-2015	Chrisy Mafnas, M.D., Santa Clara Valley Medical Center, Santa Clara, California
2015-2016	Brock Martin, M.D., University of Louisville, Louisville, Kentucky
2015-2016	Christine Louie, M.D., Veteran's Administration Medical Center, Palo Alto, California
2015-2016	Kurt Schaberg, M.D., University of California Davis, Davis, California
2016-2017	Adam Gomez, M.D., St. Joseph's Hospital and Medical Center, Phoenix, Arizona
2016-2017	Azadeh Aghel, M.D., Kaiser Permanente South San Francisco, California
2017-2018	Ling Yan, M.D., Ph.D., Kaiser Mid Atlantic Regional Laboratory, Rockville, Maryland
2017-2018	Allison Zemeck, M.D., Kaiser Permanente Downey, Downey, California
2017-2018	Greg Charville, M.D, Ph.D., Stanford University, Stanford, California
2018-2019	Alaleh Esmaeili Shandiz, M.D., University of Kentucky, Louisville, Kentucky
2018-2019	Jordan Sim, M.D., Ottawa Hospital, Ottawa, Canada
2018-2019	Shyam Sampath Raghaven, M.D. University of Virginia, Charlottesville, Virginia

2019-2020	Gregory Scott, M.D., Ph.D., Oregon Health Sciences, Portland, Oregon
2019-2020	Anne Li Chen, M.D., Albany Medical College, Albany, New York
2019-2020	Nkechi Okonkwo, M.D., The Hospital at Westlake Medical Center, Austin, Texas
2020-2021	Erna Forgo, M.D., Cleveland Clinic, Cleveland, Ohio
2020-2021	Michael Pepper, M.D., Kaiser South San Francisco, South San Francisco, California
2020-2021	Joseph Fry, M.D., Kaiser Permanente Santa Clara, Santa Clara, California
2021-2022	Steven Chirieleison, M.D., Ph.D., Stanford University, Stanford, California
2021-2022	Cindy Wang, M.D., Stanford University, Stanford, California
2021-2022	Kyra Berg, M.D., College of Physicians and Surgeons of British Columbia, Vancouver, British Columbia
2021-2022	Dana Razzano, Orlando VA Healthcare System, University of Central Florida, Orlando, Florida
2022-2023	Tolson Nichols, MD, Stanford University, Stanford, California
2022-2023	Jeenal Gordhandas, MD, Centura Health, Denver, Colorado
2022-2023	Recep Nigdelioglu, MD, Sanford Medical Center, Fargo, North Dakota
2023-2024	Eric Ollila, MD, Stanford University, Stanford California
2023-2024	Hang Yang, MD, Stanford University, Stanford, California
2023-2024	Oyewale Shiyanbola, MD, Stanford University, Stanford, California

MEDICAL STUDENT RESEARCH SCHOLARS

2012-2013	Allison Zemek, Stanford University School of Medicine, Stanford, California
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UNDERGRADUATE & HIGH SCHOOL RESEARCH ASSISTANTS

2010-2011	Sofia Liu, University of Pennsylvania, Philadelphia, Pennsylvania
2011-2012	Adita Mukund, Bellarmine College Preparatory, San Jose, California
2012-2013	Gerry Sann Rivera, Stanford University, Stanford, California
2013-2014	Jessica Li, Columbia University, New York, NY
2013-2015	Eugene Kwok, De Anza Community College, Cupertino, California
2014-2015	Jessika Baral, Mission San Jose High School, Fremont, California

VISITING SCHOLARS

2005	Takako Kiyokawa, M.D., Jikei University School of Medicine, Tokyo, Japan
2006	Thuan Cong Dang, M.D., Hue University Hospital, Hue Medical Center, Hue, Vietnam
2007	Geung Hwan Ahn, M.D., Ph.D., Sungkyunkwan University, Seoul, Korea
2007	Sharon S. Zhang, M.D., Ph.D., University of California San Diego, San Diego, California
2009	Joon Yim, M.D., Acupath Laboratories, New York, New York

2011	Esin Atik Dogan, MD, Antakya, Hatay, Turkey
2012	Vinicius Cabral, MD, Hospital de Clínicas de Porto Alegre, Porto Alegre, Brazil
2014	Justyna Szafranska, MD, Hospital de la Santa Creu i Sant Pau, Barcelona, Spain
2015	Trishe Leong, M.D., Director of Anatomic Pathology, Austin Medical Center, Melbourne, Australia
2015	Gregorio W. Pereira, M.D., Federal University of São Paulo (UNIFESP), São Paulo, Brazil
2016	Minju Lee, M.D., Samsung Medical Center, Seoul, Korea
2018	Manas R. Baisakh, M.D. Odisha, India
2018	Shing Wong, Singapore General Hospital, Singapore

PLATFORM/PLENARY SESSION PRESENTATIONS

Longacre T, Crago S, Foucar K: Clinical, Cytochemical, Flow Cytometric Immunophenotyping and DNA Content Analysis of Hematogones. Platform Presentation, International Academy of Pathology, Washington, D.C., March 1988.

Longacre T, Dressler L, Willman C: Differential Expression of Myeloid Lineage Tyrosine-Kinase Genes in Acute Myeloid Leukemia (AML). Platform Presentation, International Academy of Pathology, Washington, D.C., March 1988.

Willman C, **Longacre T**, Stewart C: Identification of a New Biological Subtype of Acute Leukemia with a Dual NK Cell - Myeloid Phenotype. Platform Presentation, International Academy of Pathology, Washington, D.C., March 1988.

Longacre TA, Fenoglio-Preiser CM: Histologic Definition of Mixed Hyperplastic-Adenomatous Polyps: A Distinct Form Of Colorectal Neoplasia. Platform Presentation, United States and Canadian Academy of Pathology, March 1989.

Baker RJ, Hildebrandt RH, Rouse RV, Hendrickson, MR, **Longacre TA**: Uterine Tumors with Sex Cord Stromal Differentiation: Evidence for True Sex Cord Differentiation. Platform Presentation at the United States and Canadian Academy of Pathology Meeting, Washington D.C., March 1998.

Shibata A, **Longacre T**, Puligandla B, Parsonnet J, Habel L: Histologic Classification Of Gastric Adenocarcinoma For Epidemiologic Research: Concordance Between Pathologists, International Epidemiological Association, Florence, Italy, September 1999.

Kambham N, Vij R, Cartwright C, **Longacre TA**, CMV Infection: A Significant Cause of Steroid-Refractory Ulcerative Colitis. Platform Presentation at the United States and Canadian Academy of Pathology Meeting, Washington D.C., March 2003.

Gilks B, Vanderhyden B, Zhu S, van de Rijn M, **Longacre T**. Distinction between Serous Borderline Tumors and Serous Carcinomas Based on mRNA Expression Profiling. Platform

Presentation at the United States and Canadian Academy of Pathology Meeting, Washington, D.C., March 2003.

Longacre T, Tazelaar H, Kempson R, Hendrickson M. Serous Tumors of Low Malignant Potential: Stanford Update. Platform Presentation at the United States and Canadian Academy of Pathology Meeting, Washington D.C., March 2003.

McKenney JM, Balzer BL, **Longacre TA**. Ovarian Serous Tumors of Low Malignant Potential with Stromal Microinvasion: A Clinicopathologic Study of 36 Cases, Platform Presentation at the United States and Canadian Academy of Pathology Meeting, Vancouver, B.C., March 2004.

McKinney JM, Balzer BL, **Longacre TA**. Histologic Patterns of Lymph Node Involvement In Women With Primary Ovarian Serous Tumors Of Low Malignant Potential (S-LMP), Platform Presentation, United States and Canadian Academy of Pathology Meeting, San Antonio, Texas, February 2005.

McKinney JM, Gilks CB, **Longacre TA**. The Classification of Extra-Ovarian Implants Associated with Ovarian Serous Tumors of Low Malignant Potential (S-LMP): clinicopathologic study of 181 cases, Platform Presentation, United States and Canadian Academy of Pathology Meeting, San Antonio, Texas, February 2005.

Liou WS, Hamilton CA, Cheung MK, Osann K, **Longacre TA**, Teng NN, Husain A, Dirbas F, Chan JK. Outcomes of women with double primary breast and ovarian carcinomas – an analysis of the SEER database. 35th Annual Meeting Society of Gynecologic Oncologists, Miami, Florida March 2005.

Hamilton CA, Cheung MK, Osann K, Husain A, Teng NN, Kapp DS, Chen LM, **Longacre TA**, Chan JK. Uterine papillary serous and clear cell histologies predict poorer survival compared to grade 3 endometrioid corpus cancer. 34th Annual Meeting of the Western Association of Gynecologic Oncologists, Santa Fe, New Mexico, June 2005.

West RB, Gilks CB, van de Rijn M, **Longacre TA**. Stromal signatures in ovarian serous tumors of low malignant potential and serous carcinoma. Platform Presentation, United States and Canadian Academy of Pathology Meeting, Atlanta, Georgia, February 2006.

Silva E, Vang R, Kurman R, Prat J, **Longacre T**. Invasive implants of serous borderline ovarian neoplasms – a multiinstitutional study. Platform Presentation, United States and Canadian Academy of Pathology Meeting, Atlanta, Georgia, February 2006.

Hazard K, **Longacre TA**. Ovarian surface epithelial neoplasms in the pediatric population. Platform Presentation, Pediatric Pathology Society Meeting, San Diego, California, March 2007.

Cuff J, **Longacre TA**. Ovarian endometrioid and clear cell carcinoma arise via different precursor lesions and have better prognosis when associated with endometriosis. Platform Presentation, United States and Canadian Academy of Pathology Meeting, Denver, Colorado, March 2009.

Mills AM, Ly A, Balzer BL, Hendrickson MR, Kempson RL, McKenney JK, **Longacre TA**. Cell cycle regulatory markers in uterine atypical leiomyoma, cellular leiomyoma, STUMP and leiomyosarcoma: immunohistochemical study of 74 cases with clinical follow-up. Platform Presentation, United States and Canadian Academy of Pathology Meeting, Washington D.C., March 2010.

Fujiwara M, Felberg A, Whittemore AS, McGuire VM, **Longacre TA**. Germline BRCA1 mutation positive ovarian cancer exhibits a distinctive highly specific phenotype. Platform Presentation, United States and Canadian Academy of Pathology Meeting, San Antonio, Texas, *International Society of Gynecological Pathologists Best Platform Presentation*, March 2011.

Moore FN, Pam, L, **Longacre TA**. Endometriosis-associated carcinomas exhibit significant site-specific differences: analysis of 396 cases. Platform Presentation, United States and Canadian Academy of Pathology Meeting, Vancouver, B.C., Canada, March 2012.

Martin B, Hazard K, **Longacre TA**. Evaluation of intestinal biopsies for pediatric enteropathy: A proposed immunohistochemical panel approach. Platform Presentation, Pediatric Pathology Society Meeting, San Diego, California, March 2013.

Martin BT, Mafnas CA, Ford JM, **Longacre TA**. Universal screening for gynecologic and colorectal cancer: A single institution experience. Platform Presentation, United States and Canadian Academy of Pathology Meeting, Boston, Massachusetts, March 2015.

Mafnas CA, Martin BT, Ford JM, **Longacre TA**. Lynch syndrome screening: discordance in MMR and germline test results. Platform Presentation, United States and Canadian Academy of Pathology Meeting, Boston, Massachusetts, March 2015.

Gomez AJ, Burton A, Steiner D, Zehnder J, **Longacre TA**. Detection of mutations in DNA polymerase ϵ (POLE) in colorectal carcinomas with intact mismatch repair proteins. Platform Presentation, United States and Canadian Academy of Pathology Meeting, *Winner of Society of Gastrointestinal Pathologists Best Platform Presentation*, El Paso, Texas, March 2017.

EXTRAMURAL PRESENTATIONS AND CONFERENCES

Workshop: Immunologic Approaches to the Diagnosis of Neoplasms. American Society of Clinical Pathology Spring Meeting, Seattle, Washington, April 1994.

The Florida Society of Pathologists' 21st Annual Anatomic Pathology Conference: Surface Epithelial Neoplasms of the Ovary and Their Differential Diagnosis, Lake Buena Vista, Florida, January 1995.

SmithKline Diagnostics: Colorectal Dysplasia and Carcinoma. San Jose, California, February 1995.

Updates in Pathology: Well Differentiated Endometrial Carcinoma: A Proposed Diagnostic Test

for Myoinvasion, University of California San Francisco, San Francisco, California, March 1995.

Workshop: Immunologic Approaches to the Diagnosis of Neoplasms. The American Society of Clinical Pathology Spring Meeting, Orlando, Florida, April 1995.

Current Concepts in Pathology: Atypical Polypoid Adenomyomas, Stanford, California, September 1995.

Workshop: Immunologic Approaches to the Diagnosis of Neoplasms. The American Society of Clinical Pathology Spring Meeting, Washington D.C., June 1996.

Co-chair and Panel Member, National Cancer Institute Breast and Ovarian Cancer Family Registry, NCI Pathology Working Group Committee Workshop, Stanford, California, September 1996.

Current Concepts in Pathology: Diagnostic Pitfalls in Gynecologic Pathology, Stanford, California, September 1996.

Moderator, Gastrointestinal Pathology Plenary Session, United States and Canadian Academy of Pathology, March 1997.

Workshop: Immunologic Approaches to the Diagnosis of Neoplasms. The American Society of Clinical Pathology Spring Meeting, Washington, D.C., September 1998.

Current Issues in Anatomic Pathology: Diagnostic Problems in Small Bowel Biopsies, San Francisco, California, May 1999.

Workshop: Immunologic Approaches to the Diagnosis of Neoplasms. The American Society of Clinical Pathology Spring Meeting, New Orleans, Louisiana, September 1999.

Workshop: Immunologic Approaches to the Diagnosis of Neoplasms. The American Society of Clinical Pathology Spring Meeting, Las Vegas, Nevada, February 2000.

Current Issues in Anatomic Pathology: Dysplasia in Inflammatory Bowel Disease: Diagnosis and Clinical Consequences, UCSF-Stanford Course, San Francisco, California, May 2000.

Current Issues in Anatomic Pathology: Atypical Polypoid Adenomyoma/Well-Differentiated Adenocarcinoma, San Francisco, California, May 2002.

National Cancer Institute Breast and Ovarian Cancer Family Registry Steering Committee: Pathology Subcommittee, Hawaii, February 2003.

Current Issues in Anatomic Pathology: Problems in the Diagnosis of Appendiceal Epithelial Tumors and Pseudomyxoma, San Francisco, California, May 2003.

National Cancer Institute: Borderline Ovarian Tumor Consensus Workshop, Bethesda,

Maryland, August 2003.

Moderator, Gynecologic Pathology Plenary Session, United States and Canadian Academy of Pathology, Vancouver, British Columbia, Canada, March 2004.

Gynecologic Pathology Evening Specialty Conference: Strategies for Predicting Site of Origin of Problematic Glandular Proliferations in Uterine Curettings, United States and Canadian Academy of Pathology, San Antonio, Texas, March 2005.

Current Issues in Anatomic Pathology: Problems in Extra-Ovarian Serous Neoplasia, San Francisco, California, May 2005.

GI Clinical Conference: Colitis: The Pathologist's Perspective, Division of Gastroenterology, Department of Internal Medicine, Stanford University School of Medicine, Stanford, California, September 2005.

Slide Seminar: Problems in Gynecological Pathology, Department of Pathology, University of New Mexico, Albuquerque, New Mexico, October 2005.

Sixth Annual International Conference on Ovarian Cancer: Serous Tumors of Low Malignant Potential: An Update, Memorial Sloan-Kettering Cancer Center, New York, NY, November 2005.

Grand Rounds: Serous Borderline Tumors: Classification, Clinical Management and Continuing Controversies, University of Pittsburgh, Pittsburgh, Pennsylvania, November 2005.

International Society of Gynecological Pathologists: Surface Epithelial Tumors of the Ovary. Part I. Borderline Tumors – Current State of the Art: Significance of Microinvasion and Lymph Node Involvement, United States and Canadian Academy of Pathology, Atlanta, Georgia, February 2006.

Grand Rounds: Ovarian Serous Tumors of Low Malignant Potential: A Risk Model for Disease Progression, University of California, Los Angeles, Los Angeles, California, March 2006.

Current Issues in Anatomic Pathology: Problematic Glandular Proliferations in Uterine Curettings: Strategies for Predicting Site of Origin, San Francisco, California, June 2006.

Visiting Professor and Lecturer: Hereditary Diffuse Gastric Cancer Syndrome, University of Manitoba, Winnipeg, Manitoba, Canada, June 2006.

Visiting Professor and Grand Rounds Lecturer: Hereditary Diffuse Gastric Cancer Syndrome: The Gene That Binds Families Together, Memorial-Sloan Kettering Cancer Center, New York, NY, October 2006.

Thirteenth Annual Practical Pathology at Whistler: Diagnostic Problems in Uterine Curettings, Chateau Whistler, Whistler, British Columbia, Canada, February 2007.

Thirteenth Annual Practical Pathology at Whistler: Mucinous Tumors in the Ovary: Primary or Metastatic? Chateau Whistler, Whistler, British Columbia, Canada, February 2007.

Short Course, Mesenchymal Neoplasms of the Female Genital Tract, United States and Canadian Academy of Pathology, San Diego, California, March 2007

Current Issues in Anatomic Pathology, Update on GI Neuroendocrine Tumors, San Francisco, California, May 2007.

5th Asia Pacific International Academy of Pathology Congress Meeting, Serous Borderline Tumors, Singapore, May 2007.

5th Asia Pacific International Academy of Pathology Congress Meeting: Mucinous Borderline Tumors, Singapore, May 2007.

Panel Member, Interesting Case Presentations, 5th Asia Pacific International Academy of Pathology Congress Meeting, Singapore, May 2007.

Women's Cancers: Surgical Pathology, Cytology and Immunohistochemistry of Breast and Genital Tumors: Endometrial Stromal and Related Tumors, Hilton Head Island, South Carolina, June 2007.

Women's Cancers: Surgical Pathology, Cytology and Immunohistochemistry of Breast and Genital Tumors: Endometrioid and Clear Cell Tumors of the Ovary: Differential Diagnosis, Hilton Head Island, South Carolina, June 2007.

Women's Cancers: Surgical Pathology, Cytology and Immunohistochemistry of Breast and Genital Tumors: The Nonneoplastic Endometrium, Hilton Head Island, South Carolina, June 2007.

Women's Cancers: Surgical Pathology, Cytology and Immunohistochemistry of Breast and Genital Tumors: Adenocarcinoma of the Cervix: Problems and Differential Diagnosis, Hilton Head Island, South Carolina, June 2007.

Women's Cancers: Surgical Pathology, Cytology and Immunohistochemistry of Breast and Genital Tumors: Mucinous Tumors of the Ovary: From Adenoma to Carcinoma and How to Rule Out Metastasis, Hilton Head Island, South Carolina, June 2007.

Diagnostic Pathology Update: Updates In Gyn Pathology, United States and Canadian Academy of Pathology, Banff, Alberta, Canada, July 2007.

Mortimer & Harold Cohen Lecturer, Serous Epithelial Neoplasms of the Ovary: Recent Developments and Diagnostic Problems, Magee-Women's Hospital, University of Pittsburgh, Pittsburgh, Pennsylvania, October 2007.

Invited Lecturer, Kaiser Permanente Hospital, Walnut Creek, Evaluation of the Gynecologic Frozen Section: Common Pitfalls and How to Avoid Them, November 2007.

Guest Speaker, Indian Continuing Medical Education, Endocervical Adenocarcinoma: Diagnostic Problems and Special Variants, Chandigarh, India, November 2007

Guest Speaker, National Indian Academy of Pathology and Microbiology, Update on Gastrointestinal Stromal Tumors: Getting the *Gist* of GIST, Chandigarh, India, November 2007.

Guest Speaker and Panel Member, California Society of Pathologists Annual Seminar, San Francisco, California, December 2007.

Visiting Professor and Guest Lecturer, Tanta University, Tanta, Egypt, February 2008.

Guest Lecture, Kaiser Permanente Hospital, Walnut Creek, Appendiceal Neoplasms: Pseudomyxoma and Other Diagnostic Problems, March 2008.

Guest Lecture, Kaiser Permanente Hospital, Walnut Creek, Sex Cord Stromal Neoplasms of the Ovary, March 2008.

Faculty, Short Course, Mesenchymal Neoplasms of the Female Genital Tract, United States and Canadian Academy of Pathology, Denver, Colorado, March 2008.

Invited Speaker, International Society of Gynecological Pathologists: Atypical Endometrial Hyperplasia and Endometrial Intraepithelial Neoplasia: A Step Towards Constructive Dialogue, United States and Canadian Academy of Pathology, Denver, Colorado, March 2008.

Invited Speaker, Rodger C. Haggitt Gastrointestinal Pathology Society: Challenging Cases in Anal Pathology, United States and Canadian Academy of Pathology, Denver, Colorado, March 2008.

Moderator, Gynecologic Pathology Plenary Session, United States and Canadian Academy of Pathology, Denver, Colorado, March 2008.

Invited Speaker, Gynecologic Pathology Evening Specialty Conference: Strategies for Evaluating Problematic Mesenchymal Tumors in Uterine Curettings, United States and Canadian Academy of Pathology, Denver, Colorado, March 2008.

Guest Lecturer, Surgical Pathology of Neoplastic Diseases, Memorial Sloan-Kettering Cancer Center, New York, NY, May 2008.

Faculty, Diagnostic Pathology Update: Updates in Gynecologic Pathology, United States and Canadian Academy of Pathology, Maui, Hawaii, July 2008.

Invited Lecturer, Ovarian Clear Cell and Serous Carcinoma: Recent Developments, Updates in Cancer Diagnosis, Samsung Hospital, Seoul, Korea, September 2008.

Guest Speaker, XXVII International Congress of the International Academy of Pathology, Uterine Mesenchymal Tumors, Athens, Greece, October 2008.

Guest Lecturer, Serous Borderline Tumors and Atypical Polypoid Adenomyoma, Gynecologic Pathology Post-Graduate Course, Kyoto, Japan, November 2008

Invited Speaker, Top Ten Diagnoses Not To Be Missed In Ovarian Pathology, Video Tutorial, California Society of Pathologists Annual Seminar, Los Angeles, California, December 2008.

Invited Speaker, Problems in Ovarian Tumor Pathology, Walnut Creek Kaiser Permanente, Walnut Creek, California, December 2008.

Slide Seminar, Selected Problems in Pelvic Serous Carcinoma, Department of Pathology, University of Washington, Seattle, Washington, February 2009

Moderator, Gynecologic Pathology Plenary Session, United States and Canadian Academy of Pathology, Boston, Massachusetts, March 2009.

Current Issues in Anatomic Pathology, Clinical Significance of MSI, KRAS and EGFR Assays in Gastrointestinal Tumors. San Francisco, California, May 2009.

Invited Lecturer, Women's Cancers: Surgical Pathology, Cytology and Immunohistochemistry of Breast and Genital Tumors, Mauna Kea Resort, Big Island of Hawaii, June 2009.

Faculty, Diagnostic Pathology Update: Updates in Gynecological and Placental Pathology, United States and Canadian Academy of Pathology, Niagara Falls, New York, July 2009.

Invited Speaker, 7th Asia Pacific International Academy of Pathology Congress Meeting, Kerala, India, August 2009.

Invited Speaker, Mismatch Repair Protein Deficiency in Colorectal Carcinoma, California Pacific Medical Center, Department of Pathology, San Francisco, CA, December 2009

Invited Speaker, Ovarian Pathology, California Society of Pathologists Annual Seminar, San Francisco, California, December 2009.

Invited Lecturer, Ohio State University Pathology Update, Columbus Ohio, August 2010

Update on Pathology of Neuroendocrine Tumors, Neuroendocrine Tumor Patient Education Conference, Caring for Carcinoid Foundation & Stanford Cancer Center, Stanford, California, September 2010

Invited Speaker, International Gynecologic Cancer Society, Prague, Hereditary "Ovarian" Cancer: Update & Role of the Fallopian Tube, October 2010

Invited Speaker, International Academy of Pathology, Surgical Pathology: Update in Ovarian Pathology: The Concept of Pelvic Serous Carcinoma, Sao Paolo, Brazil, October 2010

Invited Speaker and Panelist, International Academy of Pathology, Surgical Pathology Case Presentation, Sao Paolo, Brazil, October 2010

Invited Speaker and Panelist, Arias Stella Society, Sao Paolo, Brazil, October 2010

Invited Speaker, Avances Recientes en Patología Quirúrgica y su Impacto Terapéutico, Hospital de Oncología, Centro Médico Nacional, Siglo XXI, Mexico City, Mexico, January 2011

Invited Speaker, International Society of Gynecological Pathologists: Clear Cell Carcinoma: Not Everything Is Always as Clear As It Seems. United States and Canadian Academy of Pathology, San Antonio, Texas, February 2011

Invited Speaker, Pacific Northwest Society of Pathology, Vancouver, BC, May 2011

Invited Speaker, The British Association of Gynaecological Pathologists, London, England, June 2011

Visiting Professor, University of Hawaii, Honolulu, Hawaii, August 2011

Guest Speaker, Hawaii Society of Pathology, Honolulu, Hawaii, August 2011

USCAP Ambassador, 6th Surgical Pathology Conference of the West African Division of the International Academy of Pathology, Abuja, Nigeria, August 2011

Faculty, Gynecologic Pathology: Gross Examination of Uterine, Fallopian Tube and Ovarian Specimens, American Association of Pathologists' Assistants' 37th Annual Continuing Education and Business Conference, San Francisco, California, August 2011

Neuroendocrine Tumor Patient Education Conference, Caring for Carcinoid Foundation & Stanford Cancer Center, Stanford, California, September 2011

Invited Speaker, Oregon Pathologists Association, Portland, Oregon, September 2011

Faculty, Short Course, Hereditary Gynecologic Cancer Syndromes, American Society for Clinical Pathology, Las Vegas, Nevada, October 2011.

Invited Speaker, Update in GI Pathology, Department of Pathology, Tata Memorial Hospital, Mumbai, India, November 2011

Invited Speaker and Panel Member, Diagnostic Problems in Surgical Pathology, California Society of Pathologists Annual Seminar, San Francisco, California, December 2011.

Molecular Advances in Gynecologic Pathology: Screening for Hereditary Cancer Syndromes,

Memorial Sloan-Kettering Grand Rounds, New York, NY, March 2012

Biobanking for Clinical Care in the Molecular Era, ADASP, United States and Canadian Academy of Pathology, Vancouver, British of Columbia, Canada, March 2012.

Moderator, Gynecologic and Oncology Plenary Session, March 2012, United States and Canadian Academy of Pathology, Vancouver, British of Columbia, Canada, March 2012

Faculty, Short Course, Glandular Lesions of the Cervix: An Integrated Cytologic and Histologic Approach, United States and Canadian Academy of Pathology, Vancouver, British of Columbia, Canada, March 2012.

Invited Speaker and Panel Member, Surgical Pathology Specialty Conference, United States and Canadian Academy of Pathology, Vancouver, British of Columbia, Canada, March 2012.

Molecular Advances in Gynecologic Pathology: Screening for Hereditary Cancer Syndromes, Yale Pathology Grand Rounds, New Haven, Connecticut, April 2012

Invited Lecturer, Borderline Tumors of the Ovary, New York Pathology Society, New York, NY, May 2012

Invited Speaker, Mexico Society of Pathology Annual Meeting, Gynecologic Pathology Long Course, Queretaro, Mexico, May 2012

Invited Speaker, Problems in Anal Pathology, Stars in the Mountains, The Colorado Society of Clinical Pathologists, Vail, Colorado, July 2012

Invited Speaker, Hereditary Cancer Syndromes, Stars in the Mountains, The Colorado Society of Clinical Pathologists, Vail, Colorado, July 2012

Council Meeting, International Society of Gynecological Pathologists, WHO Classification of Gynecologic Tumors, New York, New York, August 2012

Faculty, 2012 International Academy of Pathology Congress, Cape Town, South Africa, October 2012

Invited Speaker and Panel Member, Gynecologic Pathology Slide Seminar, Cape Town, South Africa, October 2012

Invited Speaker, GI Polyps, California Society of Pathologists Annual Seminar, San Francisco, California, December 2012

Invited Speaker, Polyps and Polyposis Syndromes, Kaiser Northern California, Webinar, Stanford, California, January 2013

Invited Speaker, Molecular Medicine TriConference, San Francisco, California, February 2013

Course Instructor, Morphologic Phenotype(s) of BRCA1/2 Breast and Ovarian Cancer: Implications for Screening, American Society of Clinical Pathology, Rancho Mirage, California, February 2013

Faculty, Short Course, Glandular Lesions of the Cervix: An Integrated Cytologic and Histologic Approach, United States and Canadian Academy of Pathology, Baltimore, Maryland, March 2013

Invited Speaker, Current Issues in Surgical Pathology, University of Texas Southwestern, Dallas, Texas, April 2013

Invited Speaker, British Association of Gynecological Pathologists, 10th Annual Meeting, London, United Kingdom, June 2013

Consensus Group, Tumors of the Female Genital Tract, World Health Organization, Lyon, France, June 2013

Lecturer, Endometrial Carcinoma: Diagnosis and Histologic Subtypes, Kaiser Southern California Webinar, August 2013

Lecturer, Inflammatory Bowel Disease and the Diagnosis of Dysplasia, Kaiser Northern California Webinar, August 2013

Lecturer, Endometrial Carcinoma: Diagnosis and Histologic Subtypes, Kaiser Northern California Webinar, September 2013

Visiting Professor, University of Hawaii, Honolulu, Hawaii, August 2013

Invited Speaker, Hawaii Society of Pathology, Honolulu, Hawaii, August 2013

Short Course, American Society for Clinical Pathology, Chicago, Illinois, September 2013

Invited Lecturer, Ohio State University Pathology Update, Columbus Ohio, October 2013

Short Course, College of American Pathologists, Orlando, Florida, October 2013

Invited Lecturer, Brazilian Society of Pathology, 29th Congress, Florianopolis, Brazil, November 2013

Invited Lecturer, Ovarian Serous Borderline Tumors & Low-Grade Serous Carcinoma, Kaiser Northern California Webinar, November 2013

Lecturer, Pancreatic Cystic Lesions, Kaiser Northern California Webinar, December 2013

Short Course, Surgical Pathology of the Female Genital Tract in Pregnancy, California Society

of Pathologists Annual Seminar, San Francisco, California, December 2013

Invited Lecturer, Ovarian Serous Borderline Tumors & Low-Grade Serous Carcinoma, Kaiser Southern California Webinar, January 2014.

Invited Speaker, Molecular Medicine Tri Conference, Moscone Convention Center, San Francisco, California, February 2014

Invited Lecturer, Annual Gynecologic Pathology/Oncology Conference, Detroit, Michigan, February 2014

Faculty, Short Course, Glandular Lesions of the Cervix: An Integrated Cytologic and Histologic Approach, United States and Canadian Academy of Pathology, San Diego, California, March 2014

Faculty, Short Course, Hereditary Gynecologic Cancer Syndromes, United States and Canadian Academy of Pathology, San Diego, California, March 2014

Panelist, Hot Topics in Gynecological Pathology, United States and Canadian Academy of Pathology, San Diego, California, March 2014

Invited Lecturer, USCAP Practical Pathology Seminar, 2014, New York, New York, May 2014

Presidential Guest Lecture, Western Association of Gynecologic Oncologists, Tahoe, California, June 2014

Invited Lecturer, Florida Society of Pathologists, Palm Beach, Florida, July 2014

Faculty, Short Course, College of American Pathologists, Chicago, Illinois, September 2014

Visiting Professor, Birmingham, England, September 2014

Co-Chair and Invited Lecturer, Updates on Vulvar and Anal Pathology, International Academy of Pathology, Bangkok, Thailand, October 2014

Short Course, Endocervical Adenocarcinoma: An Integrative Cytologic and Histologic Approach, California Society of Pathologists Annual Seminar, San Francisco, California, December 2014

Grand Rounds, New York University, New York City, New York, December 2014

Faculty, Short Course, Hereditary Gynecologic Cancer Syndromes, United States and Canadian Academy of Pathology, Boston, Massachusetts, March 2015

Invited Speaker, International Society of Gynecological Pathologists: Vulvar and Anal Intraepithelial neoplasia. Diagnosis, Nomenclature and Ancillary Studies, United States and

Canadian Academy of Pathology, Boston, Massachusetts, March 2015

Invited Speaker, Gynecological Pathology Evening Panel, United States and Canadian Academy of Pathology, Boston, Massachusetts, March 2015

Invited Speaker, Association of Directors of Anatomic and Surgical Pathology, Boston, Massachusetts, March 2015

Invited Lecturer, Endocervical Glandular Lesions: Histology, Kaiser Northern California Webinar, April 2015

Guest Lecturer, Houston Society of Clinical Pathology Spring Seminar, Houston, Texas, April 2015

William T Hill Lectureship, Department of Pathology, Baylor College of Medicine, Houston, Texas, April, 2015

Invited Lecturer, Scientific Symposiums International, Celebrating the Illustrious Career of Dr. Richard L. Kempson: Surgical Pathology of the Breast, Female Genital Tract, Head & Neck and Lung, Big Island of Hawaii, July 2015

Invited Lecturer, American Society of Clinical Pathology, Pathology Update: State-of-the-Art Diagnostic Surgical Pathology, Las Vegas, Nevada, July 2015

Invited Lecturer, Pacific Northwest Society of Pathologists, Vancouver, British Columbia, Canada, September 2015.

Faculty, Short Course, American Society of Clinical Pathologists, Long Beach, California, October 2015

Faculty, Short Course, College of American Pathologists, Nashville, Tennessee, October 2015

Grand Rounds, Weill Cornell Medical Center, New York, New York, November 2015

Invited Lecturer, Gynaecological Pathology – Putting Virtual Microscopy to the Test, The Royal College of Physicians, London, UK, January 2016

Invited Lecturer, MUSC Pathology Symposia, Charlottesville, South Carolina, February 2016.

Faculty, Short Course, Hereditary Gynecologic Cancer Syndromes, United States and Canadian Academy of Pathology, Seattle, Washington, March 2016

Presenter, Mismatch Repair Protein and Microsatellite Instability Testing in Gynecologic Cancer, Biomarkers in Endometrial Cancer Subcommittee, International Society of Gynecologic Pathologists, United States and Canadian Academy of Pathology, Seattle, Washington, March

2016

Key Note Speaker, Lynch Syndrome Testing in the Female Genital Tract, Berlin, Germany, May 2016.

Grand Rounds Speaker, Lynch Syndrome in the Female Genital Tract: Diagnostic Tests and Pitfalls, University of Tubingen, Tubingen, Germany, May 2016.

Invited Lecturer, American Society of Clinical Pathology, Pathology Update, Washington, DC, July 2016

Organizer, Scientific Symposiums International, 2nd Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter: Surgical Pathology and Cytopathology of the GI and GU Tracts, Kauai, Hawaii, July 2016

Speaker, Consultations in Anal and Perianal Pathology...And You Thought Colitis Was a Pain in the..., Scientific Symposiums International, 2nd Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter: Surgical Pathology and Cytopathology of the GI and GU Tracts, Kauai, Hawaii, July 2016

Speaker, What if it's Not 'Jist' a GIST? Scientific Symposiums International, 2nd Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter: Surgical Pathology and Cytopathology of the GI and GU Tracts, Kauai, Hawaii, July 2016

Speaker, Diarrhea and Belly Pain: The Not So Usual Suspects, Scientific Symposiums International, 2nd Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter: Surgical Pathology and Cytopathology of the GI and GU Tracts, Kauai, Hawaii, July 2016

Speaker, The Survivor's Guide to Appendiceal Mucinous Tumors, Scientific Symposiums International, 2nd Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter: Surgical Pathology and Cytopathology of the GI and GU Tracts, Kauai, Hawaii, July 2016

Speaker, Caring for Carcinoid: The Problem Cases, Scientific Symposiums International, 2nd Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter: Surgical Pathology and Cytopathology of the GI and GU Tract, Kauai, Hawaii, July 2016

Visiting Professor, University of Hawaii, Honolulu, Hawaii, July 2016.

Invited Speaker, Hawaii Society of Pathology, Honolulu, Hawaii, July 2016

Invited Lecturer, Endometrial Hyperplasia: Pre- and Post-Treatment, Kaiser Northern California Webinar, August 2016.

Invited Speaker, Update on Endometrial Cancer Biomarker Testing, Cancer Biomarkers Conference, Houston Methodist Department of Pathology, Houston, Texas, September 2016

Faculty, Short Course: The Role of the Second Opinion in Surgical Pathology: Just the FAQs Ma'am. American Society of Clinical Pathology, Las Vegas, Nevada, September 2016

Faculty, Short Course: Pancreatic Quandaries: How to Handle Small Specimens – A Cytologic/Histologic Approach, American Society of Clinical Pathology, Las Vegas, Nevada, September 2016

Faculty, Short Course: Pseudoneoplastic Lesions and Mimic in the Endometrium: Navigating a Potentially Treacherous Landscape, American Society of Clinical Pathology Las Vegas, Nevada, September 2016

Arthur Purdy Stout Lecturer, Lynch Screening in the Female Genital Tract: Where Are We Now & Where Are We Headed, American Society of Clinical Pathology, Las Vegas, Nevada September 2016

Invited Speaker, Challenging Cases in Surgical Pathology, XXXI International Congress of the International Academy of Pathology & 28th Congress of the European Society of Pathology, Cologne, Germany, September 2016

Plenary Lecture, Frontiers in Pathology, University of Michigan, Ann Arbor, Michigan, October 2016

Invited Speaker, South Carolina Surgical Pathology Conference, Greenville, South Carolina, November 2016

Keynote Speaker, Melbourne Gynecologic Pathology Seminar, Melbourne, Australia, November 2016

Invited Short Course Lecturer, Uterine Mesenchymal Lesions, California Society of Pathologists Annual Seminar, San Francisco, California, December 2016

Invited Speaker: Arthur Purdy Stout Society, United States and Canadian Academy of Pathology, San Antonio, Texas, March 2017

William M. Shelley Memorial Lecture, John Hopkins Department of Pathology, John Hopkins Medical School, Baltimore, Maryland, April 2017

Organizer & Speaker, Scientific Symposiums International, 3rd Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter: The Next Generation. Surgical Pathology of the Breast, GI and GYN Tracts, Maui, Hawaii, July 2017

Invited Lecturer, American Society of Clinical Pathology, Pathology Update, Las Vegas,

Nevada, July 2017

Invited Lecturer, Austrian Society of Pathology, Velden, Austria, September 2017

Invited Faculty, Mesenchymal Lesions of the Uterus, USCAP Interactive Learning Center, Palm Springs, California, September 2017.

Invited Speaker, Pathology Research Lecture Series, University of California, San Diego, San Diego, California, February 2018.

Invited Speaker, Napa Valley Pathology Conference, Napa, California, May 2018

Invited Speaker, Great Lakes Pathology Conference – Traverse City, Michigan, June 2018

Organizer, Scientific Symposiums International, 4th Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter: Timely Updates in Lung, Head & Neck, & Skin, Maui, Hawaii, July 2018

Invited Speaker, Hawaii Society of Pathology, Honolulu, Hawaii, July 2018

Invited Lecturer, Gynecologic Tract Frozen Section Diagnoses, Kaiser Northern California Webinar, August 2018

Invited Speaker, Third Cancer Biomarker Conference (CBCIII), Houston Methodist Research Institute Auditorium, September 2018

Invited Faculty, Mesenchymal Lesions of the Female Gynecologic Tract, USCAP Interactive Learning Center, Palm Springs, California, September 2018.

Short Course, Serous Epithelial Tumors in the Female Genital Tract. College of American Pathologists, Chicago, Illinois, October 2018

Short Course, Endocervical Carcinoma. College of American Pathologists, Chicago, Illinois, October 2018

Invited Speaker, Hawaii Pathology Conference, Maui, Hawaii, October 2018

Invited Speaker, Arab Division of the International Academy of Pathology, The XXXII Congress of the International Academy of Pathology, King Hussein Bin Talal Convention Centre, Dead Sea, Jordan, October 2018.

Invited Speaker, Pathology Societies Federation of Turkey, Ankara, Turkey, October 2018

Invited Speaker, Arizona Society of Pathology, Phoenix, Arizona, November 2018

Squamous Lesions of the Lower Female Genital Tract, On Demand Webcast for the American Society of Clinical Pathologists, November 2018

Invited Speaker, Saturday Case Seminar, California Society of Pathologists Annual Seminar, San Francisco, California, December 2018.

Invited Faculty, Mighty Women of Pathology: Update in Surgical Pathology, Maui, Hawaii, January 2019.

Invited Speaker, Diagnostic Advances in Surgical Pathology and Cytopathology, University of San Diego, San Diego, California, April 2019

Invited Speaker, British Association of Gynaecological Pathologists Annual Meeting, London, England, June 2019

Organizer and Speaker, Scientific Symposiums International, 5th Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter, Kona, Hawaii, July 2019

Invited Speaker, Colorado Society of Clinical Pathologists, Stars in the Mountains, Vail, Colorado, July 2019

Grand Rounds Speaker, Oregon Health Science University, Portland, Oregon, September 2019

Invited Speaker, DICER Syndrome, 31st European Congress of Pathology, Nice, France, September 2019

Invited Faculty, Mesenchymal Lesions of the Female Gynecologic Tract, USCAP Interactive Learning Center, Palm Springs, California, September 2019

Invited Speaker, Midwestern Pathology Conference, October 2019

Invited Speaker, Cancer Biomarkers Conference IV, Saddleback, New Jersey, October 2019

Invited Speaker, 11th Asia Pacific IAP Congress, Hefei, Anhui province, China, October 2019

Invited Faculty, Mesenchymal Lesions of the Gastrointestinal Tract, USCAP Interactive Learning Center, Palm Springs, California, October 2019

Short Course, Serous Epithelial Tumors in the Female Genital Tract. College of American Pathologists, Orlando, Florida, October 2019

Short Course, Endocervical Carcinoma. College of American Pathologists, Orlando, Florida, October 2019

Guest Speaker, John Hopkins Continuing Education Course, Baltimore, Maryland, October 2019

Invited Speaker, Pacific Northwest Society of Pathology, Portland, Oregon, October 2019

Invited Speaker, British Division of the International Academy of Pathology, London, England, November 2019

Short Course, Ovarian Epithelial Tumors: New Developments. California Society of Pathologists, San Francisco, California, December 2019

Invited Speaker, Gastrointestinal Pathology Society, USCAP, Los Angeles, California, March 2020

Short Course, Serous Epithelial Tumors in the Female Genital Tract. College of American Pathologists, October 2020

Short Course, Endocervical Carcinoma. College of American Pathologists, October 2020

Invited Speaker, Indiana Association of Pathologists, Indianapolis, Indiana, May 2021.

Invited Speaker, International Society of Gynecologic Pathologists, May 2021

Invited Speaker, Colorado Society of Clinical Pathologists, Stars in the Mountains, Vail, Colorado, July 2021

Organizer and Speaker, Scientific Symposiums International, 6th Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter, Kona Hawaii, July 2021

Invited Speaker, Cancer Biomarkers Conference V, Biomarkers in Gynecologic Malignancies: Established & Emerging, Jackson, Mississippi, September 2021

Course Director and Faculty, College of American Pathologists, Gynecologic Pathology Full Day Course, Chicago, Illinois, September 2021

Invited Faculty, Gynecologic Pathology: Navigating Histologic Mimics and other Diagnostic Pitfalls, USCAP Interactive Learning Center, Palm Springs, California, October 2021

Invited Faculty, Arthur Purdy Stout Society, USCAP Interactive Learning Center, Palm Springs, California, October 2021

Invited Speaker, California Society of Pathologists, San Francisco, California, December 2021

Guest Speaker, Mayo Clinic Surgical Pathology Update, Phoenix, Arizona, January 2022.

Guest Speaker, Canadian Anatomic and Molecular Pathology Conference, May 2022.

Organizer and Speaker, Scientific Symposiums International, 7th Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter, Kona, Hawaii, July 2022.

Invited Speaker, International Society of Gynecologic Pathologists, November 2022

Invited Speaker, California Society of Pathologists, San Francisco, California, December 2022

Invited Speaker, International Society of Gynecologic Pathologists, New Orleans, Louisiana March 2023

Organizer and Speaker, Scientific Symposiums International, 8th Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter, Kona, Hawaii, July 2023

Invited Speaker, Gastrointestinal Pathology, American Society of Clinical Pathologists, Long Beach, California, October, 2023

Invited Speaker, Mismatch Repair Analysis in Neoplasms, Kaiser Webinar, November, 2023

Invited Speaker, Florida Society of Pathologists, Florida Society of Pathologists 50th Annual Pathology Conference, Orlando, Florida, February 16-18, 2024.

Invited Speaker, USCAP and ISGYP Co-branded Interactive Microscopy Course, Palm Springs, California, February 21-24, 2024

Invited Speaker, 65th Annual South Bay Pathology Society Spring Meeting, Menlo Park, California, April, 2024

Invited Speaker, The 16th Annual World Cancer Congress, Budapest, Hungary, June 2024.

Invited Speaker, Colorado Society of Clinical Pathologists, Stars in the Mountains, Vail, Colorado, July 2024

Organizer and Speaker, Scientific Symposiums International, 9th Annual Dr. Richard L. Kempson Diagnostic Pathology Course: Making Diagnoses That Matter, Kona, Hawaii, July 2024

Invited Speaker, Montana Pathologists Society Annual Meeting, Bozeman, Montana, September 2024

Invited Speaker, Luminaries in Pathology, United States and Canadian Academy of Pathology, Boston, Massachusetts, March 2025

PEER-REVIEWED JOURNAL ARTICLES

1. **Longacre TA**, Bartow SA: A correlative morphologic study of breast and endometrium in the menstrual cycle. *Am J Surg Pathol* 1986; 10: 382-393.
2. **Longacre TA**, Foucar K, Crago S, Chen I-M, Griffith B, Dressler L, McConnell TS, Duncan M, Gribb J: Hematogones: A multiparameter analysis of bone marrow precursor cells. *Blood* 1989; 73:543-552.
3. **Longacre T**, Foucar K, Koster F, Burgdorf W: Atypical cutaneous lymphoproliferative disorder resembling mycosis fungoides in AIDS: Report of a case with concurrent Kaposi's sarcoma. *A J Dermatopathol* 1989; 11:451-456.
4. **Longacre TA**, Listrom MB, Spigel JH, Willman CL, Dressler L, Clark D: Aggressive jejunal lymphoma of large granular lymphocytes: Immunohistochemical, ultrastructural, molecular and DNA content analysis. *Am J Clin Pathol* 1990; 93:124-132.
5. **Longacre TA**, Fenoglio-Preiser CM: Mixed hyperplastic adenomatous polyps/serrated adenomas: A distinct form of colorectal neoplasia. *Am J Surg Pathol* 1990; 14:524-537.
6. Willman CL, Stewart CC, **Longacre TA**, Head DR, Habbersett R, Ziegler SF, Perlmutter RM: Expression of the c-fgr and hck protein-tyrosine kinases in acute myeloid leukemic blasts is associated with early commitment and differentiation events in the monocytic and granulocytic lineages. *Blood* 1991; 77:726-734.
7. Smoller BR, **Longacre TA**, Warnke RA: Ki-1 (CD30) expression in differentiation of lymphomatoid papulosis from arthropod bite reactions. *Modern Pathology* 1992; 5:492-496.
8. **Longacre TA**, Smoller BR: Leukemia cutis. Analysis of 50 biopsy-proven cases with emphasis on occurrence in myelodysplastic syndromes. *Am J Clin Pathol* 1993; 100:276-284.
9. **Longacre TA**, Smoller BR, Rouse RV: Atypical fibroxanthoma: Multiple immunohistologic profiles. *Am J Surg Pathol* 1993; 17:1199-1209.
10. Davis RE, **Longacre TA**, Cornbleet PJ: Hematogones in the bone marrow of adults: Immunophenotypic features, clinical settings and differential diagnosis. *Am J Clin Pathol*, 1994; 102:202-211.
11. **Longacre TA**, Chung MH, Jensen DN, Hendrickson MR: Proposed criteria for the diagnosis of well differentiated endometrial carcinoma: a diagnostic test for myoinvasion. *Am J Surg Pathol*, 1995; 19:371-406.
12. Wallace ML, **Longacre TA**, Smoller BR: Estrogen and progesterone receptors and BRST-2 fail to distinguish metastatic breast carcinoma from eccrine neoplasms. *Modern Pathol*, 1995; 8:897-901.
13. Rouse RV, Soetikno RM, Baker RJ, Barnard IC, Triadafilopoulos G, **Longacre TA**:

Esophageal submucosal gland duct adenoma. *Am J Surg Pathol*, 1995; 19:1191-1196.

14. O'Hanlan K, Kargas S, Schreiber M, Hendrickson M, **Longacre T**, Burrs D: Ovarian carcinoma metastases to gastrointestinal tract appear to spread like colon carcinoma to mesenteric lymph nodes: Implications for surgical resection. *Gynecol Oncol*, 1995; 59:200-206.
15. **Longacre TA**, Chung MH, Rouse RV, Hendrickson MR: Atypical polypoid adenomyofibromas (atypical polypoid adenomyomas) of the uterus. A clinicopathologic study of 55 cases. *Am J Surg Pathol*, 1996; 20:1-20.
16. Soslow RA, Chung MH, Rouse RV, Hendrickson MR, **Longacre TA**: Atypical polypoid adenomyofibroma (APA) versus well differentiated endometrial carcinoma with prominent stromal matrix: an immunohistochemical study. *Int J Gynecol Pathol*, 1996; 15:209-216.
17. **Longacre TA**, O'Hanlan K, Hendrickson, MR: Adenoid cystic carcinoma of the submandibular gland with symptomatic ovarian metastases. *Int J Gynecol Pathol*, 1996; 15:349-355.
18. Soslow RA, Rouse RV, Hendrickson MR, Silva EG, **Longacre TA**: Transitional cell neoplasms of the ovary and urinary bladder: a comparative immunohistochemical analysis. *Int J Gynecol Pathol*, 1996; 15:257-265.
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CORRESPONDENCE

1. Lennerz JK, Pantanowitz L, Amin M, Eltoum I-E, Hameed M, Kalof A, Khanafshar E, Kunju L, Lazenby A, Montone K, Otis C, Reid M, Staats P, Whitney-Miller C, Abendroth C, Manju A, Birdsong G, Bleiweiss I, Bronner M, Chapman J, Cipriani N, de la Roza G, Esposito M, Fadare O, Ferrer K, Fletcher C, Frishberg D, Garcia F, Geldenhuys L, Gill R, Gui D, Halat S, Hameed O, Hornick J, Huber A, Jain D, Jhala N, Jorda M, Jorns J, Kaplan J, Khalifa M, Khan A, Kim G, Lee E, LiVolsi V, **Longacre T**, Magi-Galluzzi C, McCall S, McPhaul L, Mehta V, Merzianu M, Miller S, Molberg K, Moreira A, Naini B, Nose V, O'Toole K, Picken M, Prieto V, Pullman J, Quick C, Reynolds J, Rosenberg A, Schnitt S, Schwartz M, Sekosan M, Smith MT, Sohani A, Stowman A, Vanguri V, Wang B, Watts J, Wei S, Whitney K, Younes M, Zee S, Bracamonte E. Ensuring remote diagnostics for pathologists: an open letter to the US Congress. In Press, Nature Medicine 2022

ABSTRACTS (NOT PUBLISHED ELSEWHERE)

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8. Rogers W, Mulligan AM, Bane A, West D, Andrulis I, Whittemore A3, Senie R, O'Malley FP, John EM, **Longacre TA**. Histologic characterization of *in situ* carcinoma in BRCA1 and BRCA2 mutation carriers without invasive disease: a population-based study, *Mod Pathol* 2008;21:52A.
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12. DiMaio MA, Pai RK, **Longacre TA**. PAX8 differentiates gastrointestinal carcinomas from mucinous carcinomas of the ovary, but not mucinous carcinomas arising in ovarian teratomas. *Mod Pathol* 2012;25:266A.
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LETTERS

1. Lennerz JK, Pantanowitz L, Amin MB, Eltoum I, Hameed, MR, Kalof AN, Khanafshar E, Kunju, L. P., Lazenby, A. J., Montone, K. T., Otis, C. N., Reid, M. D., Staats, P. N., Whitney-Miller, C. L., Abendroth, C. S., Aron, M., Birdsong, G. G., Bleiweiss, I. J., Bronner, M. P., Chapman, J., Cipriani, N. A., de la Roza, G., Esposito, M. J., Fadare, O., Ferrer, K., Fletcher, C. D., Frishberg, D. P., Garcia, F. U., Geldenhuys, L., Gill, R. M., Gui, D., Halat, S., Hameed, O., Hornick, J. L., Huber, A. R., Jain, D., Jhala, N., Jorda, M., Jorns, J. M., Kaplan, J., Khalifa, M. A., Khan, A., Kim, G. E., Lee, E. Y., LiVolsi, V. A., Longacre, T., Magi-Galluzzi, C., McCall, S. J., McPhaul, L., Mehta, V., Merzianu, M., Miller, S. B., Molberg, K. H., Moreira, A. L., Naini, B. V., Nose, V., O'Toole, K., Picken, M., Prieto, V. G., Pullman, J. M., Quick, C. M., Reynolds, J. P., Rosenberg, A. E., Schnitt, S. J., Schwartz, M. R., Sekosan, M., Smith, M. T., Sohani, A., Stowman, A., Vanguri, V. K., Wang B, Watts JC, Wei S, Whitney K, Younes M, Zee S, Bracamonte ER. Ensuring remote diagnostics for pathologists: an open letter to the US Congress. *Nat Med* 2022 Oct 20. doi: 10.1038/s41591-022-02040-6. Online ahead of print

FORMER GRANT SUPPORT

Co-Investigator, Epidemiology of Helicobacter Pylori Transmission, NIH Grant R01 AI042801, for the period 03/01/06 – 02/28/11, 1%

Co-Investigator, Epigenetic Changes and Phenotype-Specific Therapeutic Strategies in Breast Cancer, University of Utah, R01 GM085601-01, for the period 07/01/08 – 6/30/12, 5%

Principle Investigator, Caring for Carcinoid Foundation Neuroendocrine Tumor Biospecimen Consortium, Caring for Carcinoid Foundation, for the period 01/01/09 - 12/31/10, 1%

Co-Investigator, Molecular Diagnosis of Human GI Cancers for Surgical Tumor Margin Assessment Using Ambient Mass Spectrometry, Stanford Hospital& Clinics: Innovation Fund Award, for the period 11/15/12 – 11/14/13, 2%

Co-Principal Investigator, A Tissue Microarray for Neuroendocrine Tumors, Developmental Cancer Research Award, Stanford Cancer Institute, for the period 11/1/13 – 11/1/14, 5%

Co-Investigator, Prognostic & Therapeutic Advances through Systematic Immuno-Genomic Characterization of Carcinoid Tumors, Caring for Carcinoid Foundation, \$100,000.00, for the period 04/06/2015 to 04/05/2016, 1%.

Co-Investigator, Molecularly Targeted Ultrasound in Ovarian Cancer NIH 5R01CA21193204, \$3,522.44, for the period of 5/1/2019-4/30/2023, 3%

Co-Investigator, Human Tumor Atlas Network, Precancer Atlas of Familial Adenomatous Polyposis NIH 1U2CCA233311-01, \$49,379.13 for the period of 9/30/2018-6/30/23, 4%

Co-Investigator, Stanford Tissue Mapping Center, NIH 5U54HG010426-03, 7,377.50 for the period of 9/30/2018-6/30/2022

Co-Investigator, Molecular Imaging Methods for the Detection of Pancreatic Ductal Adenocarcinoma NIH 1U01CA21002001A, \$4,676,729, for the period 5/1/2017-4/30/2023, 1%.

CURRENT GRANT SUPPORT

Stanford Cancer Institute, NIH 2P30CA124435-14, \$40,215,947, for the period 6/4/2007-5/31/2127, 10%

3/16/2024

EXHIBIT B

Newsome, Tamara

EXHIBIT B**Histology Slides from Holy Cross Hospital**

Sample No.	Description
1	S15-2514 FS1 H&E
2	S15-2514 A2 H&E
3	S15-2514 A3 H&E
4	S15-2514 A4 H&E
5	S15-2514 A5 H&E
6	S15-2514 A6 H&E
7	S15-2514 A7 H&E
8	S15-2514 A8 H&E
9	S15-2514 A8-1 UNSTAINED
10	S15-2514 A8-2 UNSTAINED
11	S15-2514 A8-3 UNSTAINED
12	S15-2514 A8-4 UNSTAINED
13	S15-2514 A8-5 UNSTAINED
14	S15-2514 A8-6 UNSTAINED
15	S15-2514 A8-7 UNSTAINED
16	S15-2514 A8-8 UNSTAINED
17	S15-2514 A8-9 UNSTAINED
18	S15-2514 A8-10 UNSTAINED
19	S15-2514 A9 H&E
20	S15-2514 A9-1 UNSTAINED
21	S15-2514 A9-2 UNSTAINED
22	S15-2514 A9-3 UNSTAINED
23	S15-2514 A9-4 UNSTAINED
24	S15-2514 A9-5 UNSTAINED
25	S15-2514 A9-6 UNSTAINED
26	S15-2514 A9-7 UNSTAINED
27	S15-2514 A9-8 UNSTAINED
28	S15-2514 A9-9 UNSTAINED
29	S15-2514 A9-10 UNSTAINED
30	S15-2514 A10 H&E
31	S15-2514 A11 H&E
32	S15-2514 A12 H&E
33	S15-2514 A12-1 UNSTAINED
34	S15-2514 A12-2 UNSTAINED
35	S15-2514 A12-3 UNSTAINED
36	S15-2514 A12-4 UNSTAINED
37	S15-2514 A12-5 UNSTAINED
38	S15-2514 A12-6 UNSTAINED

Newsome, Tamara

Sample No.	Description
39	S15-2514 A12-7 UNSTAINED
40	S15-2514 A12-8 UNSTAINED
41	S15-2514 A12-9 UNSTAINED
42	S15-2514 A12-10 UNSTAINED
43	S15-2514 A13 H&E
44	S15-2514 A14 H&E
45	S15-2514 A15 H&E
46	S15-2514 A16 H&E
47	S15-2514 A17 H&E
48	S15-2514 A18 H&E
49	S15-2514 A19 H&E
50	S15-2514 A20 H&E
51	S15-2514 A21 H&E
52	S15-2514 B1 H&E
53	S15-2514 B2 H&E
54	S15-2514 B3 H&E
55	S15-2514 B4 H&E
56	S15-2514 B5 H&E
57	S15-2514 B6 H&E
58	S15-2514 B7 H&E
59	S15-2514 B8 H&E
60	S15-2514 B9 H&E
61	S15-2514 C H&E

Pathology Reports

S15-2514

S15-2514 Corrected

Operative Reports

03/23/2015 Operative Report

03/23/2105 Post-operative Report

Genetic Reports

Myriad Genetic Report

Additional Medical

Medical records produced by plaintiff and providers through April 2024

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Case Materials

Complaint (12/13/2018)
Amended Complaint (01/30/2019)
Plaintiff Profile Form (07/08/20)

Plaintiff Expert Reports

Daniel Clarke-Pearson, MD
John J Godleski, MD

Expert Depositions

John J Godleski, MD (03/28/24, 03/29/24, 04/19/24)

Fact Depositions

Albert Steren, MD (02/17/2021)
Daniel Francois Jr. (05/13/2021)
Ravid Garg, MD (02/04/2021)
Tamara Newsome (12/09/2020)